#### REMEDIAL INVESTIGATION REPORT OPERABLE UNIT 4: WEST SIDE AQUIFERS NAS MOFFETT FIELD, CALIFORNIA

VOLUME 4
APPENDICES D, E, and F

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Prepared by:

IT Corporation 312 Directors Drive Knoxville, Tennessee 37923

Submitted by:

The Hazardous Waste Remedial Actions Program Martin Marietta Energy Systems, Inc.
Oak Ridge, Tennessee 37831-6501

for:

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### REMEDIAL INVESTIGATION REPORT OPERABLE UNIT 4: WEST SIDE AQUIFERS

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Potentiometric Surface Contour Map, A1-Aquifer, November/December 1990

Potentiometric Surface Contour Map, A2-Aquifer, November/December 1990

Potentiometric Surface Contour Map, A1-Aquifer, March 1991

Potentiometric Surface Contour Map, A2-Aquifer, March 1991

Potentiometric Surface Contour Map, A1-Aquifer, May 1991

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- Aquifer A2	/B9-84
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Site 14 - Aquifer A1	Site 14/B14-1
- Aquifer A2	/B14-7
Results of Validated Sample Analyses	
Site CLEAN - Aquifer A1	CLEAN/BCLN-1
- Aquifer A2	/BCLN-11
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Site MEW - Aquifer A1	MEW/BMEW-1
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W8-3(C)	
W8-4(A1)	
W8-6(A1)	
W8-8(A1)	
W8-10(A2)	
W8-11(A2)	
W8-12(A2)	
CPT-(IT)	Site 8 - CPT (IT)
CPT8-3	(11)
CPT8-5	
CPT8-7	
CPT8-10	
CPT8-12	
CPT8-14	
CPT8-18	
CPT8-19	
CPT8-21	
<u>CPT - (CLEAN)</u>	Site 8 - CPT (CLEAN)
CPT-2	
CPT-4	
CPT-5	
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W9-37(A1)	
W9-38(A1)	
W9-39(B2)	
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PT9-3(A2)	
WELLS - (CLEAN)	Site 9 - Wells
	(CLEAN)
W29-1(A)	
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W89-1(A)	
W89-2(A)	
W89-11(B1)	
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WELLS - (MEW)	Site 9 - Wells
MEW 2(C)	(MEW)
MEW-2(C) MEW-3(B)	
MEW-3(B) MEW-3(C)	
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RW-9	

## VOLUME 3, APPENDIX C BORING LOGS/CPT (Continued)

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MEW-12(B)	
MEW-45(A)	
MEW-46(A)	
MEW-49(A)	
MEW-54(A)	
MEW-57(A)	
MEW-61(A)	
MEW-64(B)	
MEW-65(A)	
MEW-79(A)	
MEW-82(A)	
CPT - (IT)	Site 9 - CPT
CPT-9-5	(IT)
CPT-9-6	
CPT-9-9	
CPT-9-11	
СРТ-9-21	
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CPT-9-25	
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CPT-9-32	
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CPT-29-40

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SITE 2	GB-1 GB-2 GB-3	Site 2
SITE 3	GB-13 GB-14 GB-15 GB-16 GB-17	Site 3
SITE 4	GB-18 GB-19 GB-20	Site 4
SITE 5	GB-21 GB-22 GB-23	Site 5
SITE 6		Site 6
SITE 7	GB-24	Site 7
SITE 8	GB-25 GB-26	Site 8

# VOLUME 3, APPENDIX C (Continued) GEOPHYSICAL LOGS (Continued)

#### **FOLLOWS TAB**

SITE 9	Site 9
GB-7	
GB-8	
GB-9	
GB-10	
GB-11	
GB-12	
<b>35 12</b>	
SITE 10	Site 10
GB-27	5
GB-30	
<b>GD</b> 30	
SITE 11	Site 11
SITE 12	Site 12
SITE 13	Site 13
SITE 14	Site 14
GB-28	
SITE 15	Site 15
SITE 16	Site 16
SITE 17	Site 17
SITE 18	Site 18
SITE 19	Site 19
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#### VOLUME 4, APPENDIX D GROUNDWATER FLOW MODEL

#### FOLLOWS TAB

**MODFLOW Input** 

D, Groundwater Flow Model

**MODFLOW Output** 

SOLUTE TRANSPORT MODEL

**FOLLOWS TAB** 

Boundary Source Only, No Retardation

D, Solute Transport Model

Boundary Source Only, Retardation = 2.02 Additional Source, Without Retardation

Additional Source, Retardation = 2.02

## VOLUME 4, APPENDIX E RISK ASSESSMENT MODELS

#### **FOLLOWS TAB**

Indoor Air Modeling Concentration in Vegetables

E

#### VOLUME 4, APPENDIX F AQUIFER TESTS

#### **FOLLOWS TAB**

Westside Aquifer Test Analysis

F

# Appendix D Groundwater Flow Model and Solute Transport Model

This appendix contains input and output data for the flow model and output data for the solute transport model. Input for the flow model consists of the modular input data files. Each data file is marked for the reader's convenience. Input data are followed by the model output.

Only the output for the solute transport model is included. Four separate output files are included reflecting the conditions imposed. The output files list all input parameters before the computational results are given.

#### D.1.0 MODFLOW Model Development

For development of a three-dimensional flow model for Moffett Field, the aquifer system was divided into four horizontal layers to represent the A1-, A2-, B2-, and B3-aquifer zones as defined in Chapter 3.0 of the Remedial Investigation (RI) Report. The aquitards were simulated by varying the leakance between the appropriate aquifer zone. The input parameters for the computer code were developed from data obtained during the field investigation and from the RI/Feasibility Study (FS) reports for the Middlefield-Ellis-Whisman (MEW) Area. In the model, Layer 1 corresponds to the A1-aquifer zone, Layer 2 to the A2-aquifer zone, Layer 3 to the B2-aquifer zone, and Layer 4 to the B3-aquifer zone.

Aquifer boundary conditions were established for the bay and southern portions of the model area. General head boundaries were used for the upgradient boundary (southern) to simulate heads controlled by a piezometric surface external to the model and removed at an unknown distance. Constant head boundaries were used to simulate heads controlled by the external source or sink within the Model Area. The hydraulic heads near the bay were known to lie below sea level within the A1- and A2-aquifers and were simulated by constant head boundaries. As indicated in Chapters 3.0 and 5.0 of the RI Report, the low heads are believed to be the result of excessive water withdrawal.

The following steps were followed to develop the three-dimensional flow model for Moffett Field:

- Determine thickness of aquifer units and layer limits.
- Establish optimum finite difference size.
- Establish water transmission properties of each layer and areal heterogeneities.
- Assign constant head or river boundaries and leakance terms to salt pond/bay cell or node.
- · Determine area recharge values for Base area.
- Develop general head boundary terms for off-site hydraulic heads.
- Determine leakance terms for aquitards separating layers.
- Calibrate model against known groundwater elevation configuration.

#### D.1.1 Aquifer Zone Characteristics

Aquifer and aquitard thickness for each unit has been established from geophysical and well boring log data (IT calculation check prints, 1991a). Cumulative thicknesses of each aquifer zone were summed and plotted on isopach maps. Using the isopach maps, the thickness variations were discretized into domains of equal thickness for each model layer and then transformed into transmissivity domains for each area by multiplying by the appropriate hydraulic conductivity value.

The finite difference block size that allowed adequate resolution with a reasonable number of grid blocks was 300 by 300 feet.

Transmissivities/hydraulic conductivities for aquifer materials below the A1-aquifer at Moffett Field are poorly constrained. Pump tests have been conducted in the A1-aquifer by PRC Environmental Management, Inc. (PRC), and in the RI Report for the MEW sites by Harding Lawson Associates (HLA, 1987). The results are summarized here as Table D.1-1. Transmissivities/hydraulic conductivities for the A1 and A2 zones calculated from aquifer tests conducted during this RI are presented in Appendix F.

Table D.1-1

Hydraulic Conductivities for Aquifers at MEW<sup>a</sup>

(ft/min)

Source	<b>A</b> 1	A2	B2	В3
HLAb	0.79	0.25	2.4 x 10 <sup>-3</sup>	2.5 x 10 <sup>-3</sup>
PRC 1991	0.18	•	-	-

<sup>&</sup>lt;sup>a</sup>Please refer to Section 3.6 of the RI for comparisons between MEW, PRC, and IT aquifer

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designations. Aquifer units in this table follow IT designations.

bData from HLA derived from pump tests conducted during the MEW RI/FS in 1987/1988.

See Figure 3.6-2 for correlations between MEW and IT aquifer designations.

As discussed in Section 3.6, there are differences in aquifer definition between the MEW area and Moffett Field. These differences may be, in part, due to facies changes. Table 3.6-1 provides the comparisons between MEW, PRC, and IT designations of aquifer zones. Based on the thickness and depth to the top of the respective aquifer zones, there may be some thickening and thinning of the aquifers. From examination of this figure, it is apparent that the aquifer zone designated B1 at the MEW area corresponds with the aquifer zone designated A2 at Moffett Field.

From analysis of data available for aquifer materials in the MEW area (IT, 1991b), the aquifer materials in the A1 and A2-aquifer zones are heterogeneous. For the purpose of setting up the flow model, it was assumed, based on boring logs and pumping tests, that the aquifer is principally silty sand, cut by sand channels. Hydraulic conductivity values for these material types are provided in Table D.1-2.

Table D.1-2

Hydraulic Conductivity Assigned to Model Material Types

Soil Types	Unified Soil Classification System Symbol	K (cm/s) <sup>a</sup>	K (ft/day)
Silty sand	SM or SC	1 x 10 <sup>-5</sup> to 1.0 x 10 <sup>-4</sup>	0.028 to 0.280
Sand	SP or SW	1 x 10 <sup>-3</sup> to 1 x 10 <sup>-1</sup>	2.83 to 28.3
Gravels	GC to GM	1 x 10 <sup>-2</sup> to 1 x 10 <sup>0</sup>	28.3 to 283.0

<sup>&</sup>lt;sup>a</sup>From Freeze and Cherry, 1979.

#### D.1.1.1 A1-Aquifer Zone Hydraulic Conductivity Distribution

Hydraulic conductivity ranges from a high value of 1.6 cm/s (4535.4 ft/day) (PRC, 1991) to a low value of  $1.8 \times 10^{-3}$  cm/s (5.10 ft/day) (HLA, 1988; IT calculation sheets, 1991c). Within Site 9 values for hydraulic conductivity are on the order of 0.10 to 0.5 cm/s (283 to 1,417 ft/day) with notable outliers. Values for hydraulic conductivity from tests in the MEW Area are on the order of  $1 \times 10^{-1}$  to  $1 \times 10^{-3}$  cm/s (283 to 0.28 ft/day) (HLA, 1988), also with outliers. The A1-aquifer materials in the MEW Area are assumed to comprise poorly

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graded fine sands with  $K = 1.0 \times 10^{-2}$  cm/s (2.83 ft/day). At Moffett Field, which lies closer to the bay, the fine fractions of the sands are assumed to be more dominant with a hydraulic conductivity on the order of  $1 \times 10^{-3}$  cm/s (0.29 ft/day). Pump tests in the area of Site 9 suggest the presence of localized sand channels, and borings indicate unit thickening. Figure D-1 indicates the distribution of material types and the zones used to develop the A1-aquifer zone hydraulic conductivity model. Hydraulic conductivity in the sand channels defined in IT calculation sheets, (1991a) is assumed to be  $1 \times 10^{-1}$  cm/s. Table D.1-3 provides the values of hydraulic conductivity assigned to the zones shown in Figure D-1.

Table D.1-3

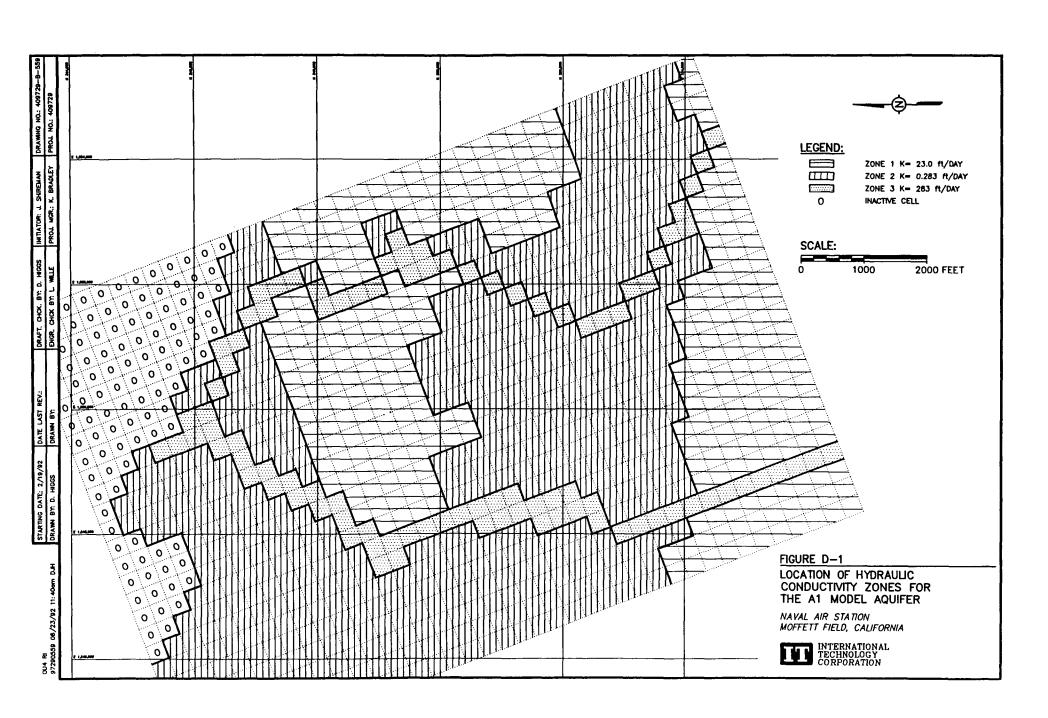
Hydraulic Conductivity Values Assigned to Model Zones for A1-Aquifer

Zone	K		
Zone 1	28.3 ft/day		
Zone 2	0.283 ft/day		
Zone 3	283 ft/day		
Vertical hydraulic conductivity A1/A2	1.78 x 10 <sup>-2</sup> ft/day		

#### D.1.1.2 A2-Aquifer Zone Hydraulic Conductivity Distribution

All aquifer model layers that are below the upper most model layer require transmissivity as input rather than hydraulic conductivity. Transmissivity was computed as the product of the thickness of aquifer material (b) and hydraulic conductivity. Hydraulic conductivity for the A2-aquifer zone was derived from pump tests conducted by HLA (1988). As shown in IT calculation sheets (1991a), the hydraulic conductivity for the A2-aquifer (MEW B1-aquifer) ranges from  $1.4 \times 10^{-3}$  to  $2.5 \times 10^{-1}$  cm/s (3.97 to 708 ft/day). There are fewer data control points for the A2-aquifer, and fewer pump tests were run; however, channels mapped for both the A1-and A2-aquifer zones appear to fall in the same general areas (see Figures 3.4-14 and 3.4-15).

Hydraulic conductivity ranges from  $1 \times 10^{-3}$  to  $1 \times 10^{-1}$  cm/s (2.83 to 28.3 ft/dy) with values around  $1 \times 10^{-2}$  cm/s occurring 8 out of 18 times (HLA, 1988, and summarized in calculations in IT calculation sheets, 1991c). The A2-aquifer zone at Moffett Field appears to be continuous with the B1-aquifer at the MEW area. These results suggest that hydraulic



conductivity distribution is similar to that of the A1-aquifer zone (compare Figure D-1 to Figure D-2). This assumption was used in developing the A2 model layer. Table D.1-4 lists transmissivity zones (derived from data presented in IT calculation sheets, 1991a).

Table D.1-4

Transmissivity Values Assigned to Model Zones for A2- Aquifer

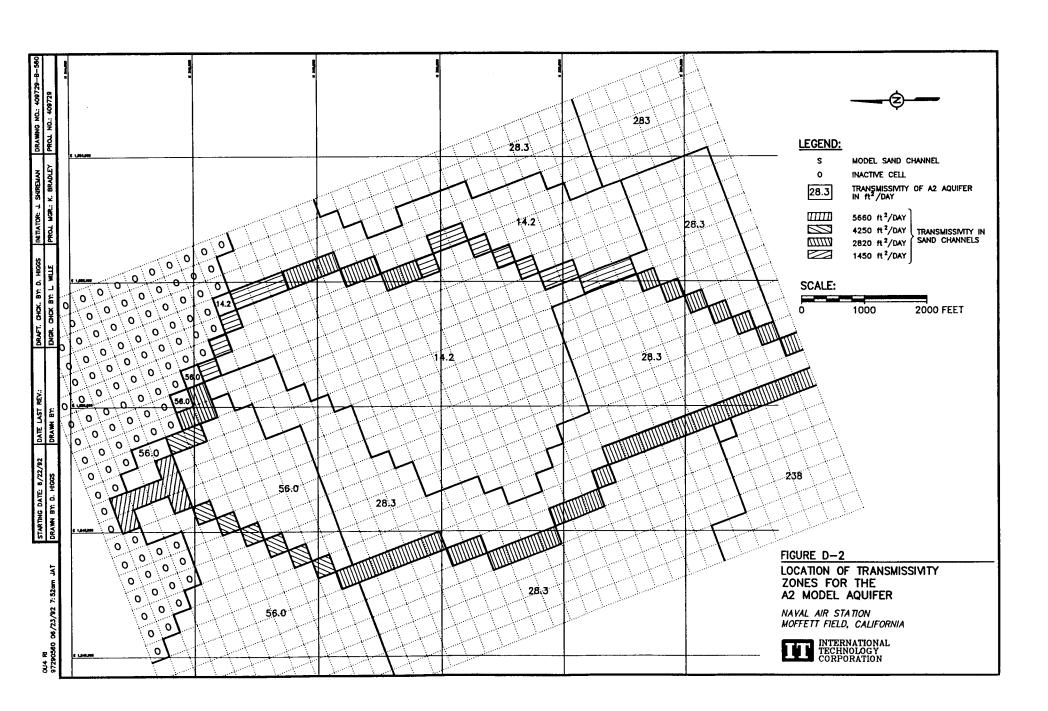
	Thickness Zones				
	$b^a = 5$	b = 10	b = 15	b = 20	
K	T (ft <sup>2</sup> /day)	T (ft <sup>2</sup> /day)	T (ft <sup>2</sup> /day)	T (ft2/day)	
K Zone 1 = 28.3 ft/day	142	283	425	566.0	
K Zone $2 = 2.83$ ft/day	14.2	28.3	42.5	56.6	
K Zone $3 = 283.0$ ft/day	1420	2830	4250	5660	

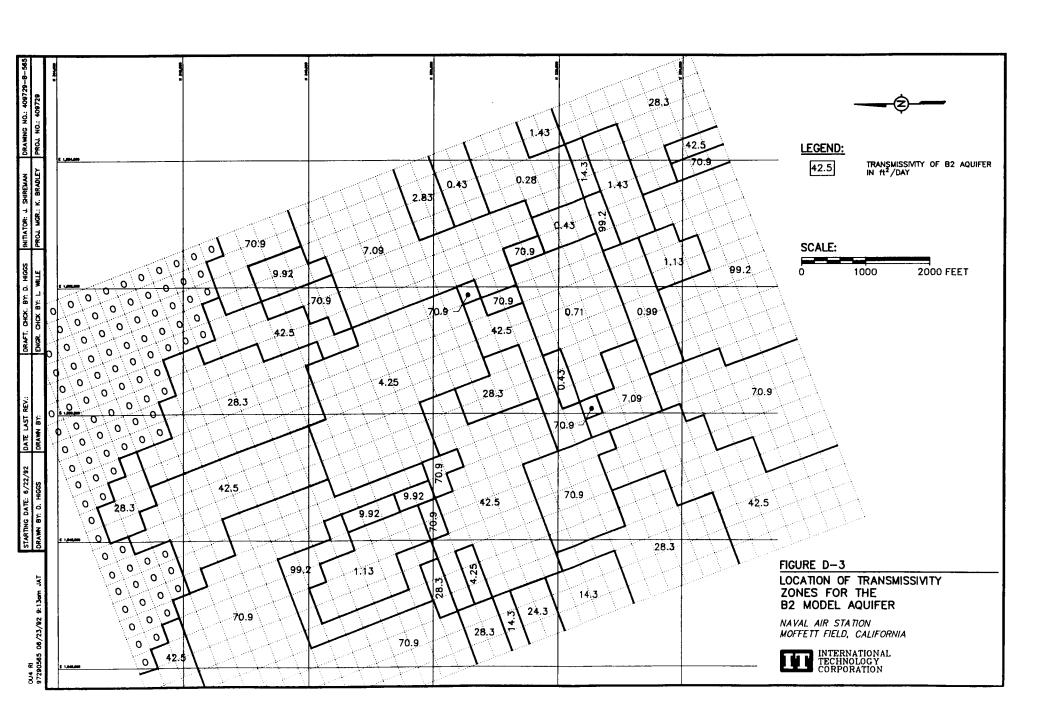
<sup>&</sup>lt;sup>a</sup>b = thickness

#### D.1.1.3 B2- and B3-Aquifer Unit Hydraulic Conductivity Distribution

Data for the B2-aquifer are limited to three monitoring wells. The mean value for the hydraulic conductivity is approximately 1 x 10<sup>-3</sup> cm/s (2.83 ft/day). The B2-aquifer is considered as confined (IT, 1991b) and therefore is treated in the same way as the A2-aquifer (i.e., the thickness and hydraulic conductivity are entered to the computer model as transmissivity). Thickness domains are obtained from IT calculation sheets (1991b). The thicknesses of individual domains and the corresponding transmissivity domains derived are provided in Table D.1-5.

Data for the B3-aquifer are limited; therefore, it was hypothesized that data from the B2-aquifer exhibits the same mean values. Transmissivity for both B2- and B3-aquifers are distributed based on thickness zones. Figures D-3 and D-4 show the location of transmissivity zones used in the flow model for the B2- and B3-aquifer zones, respectively.





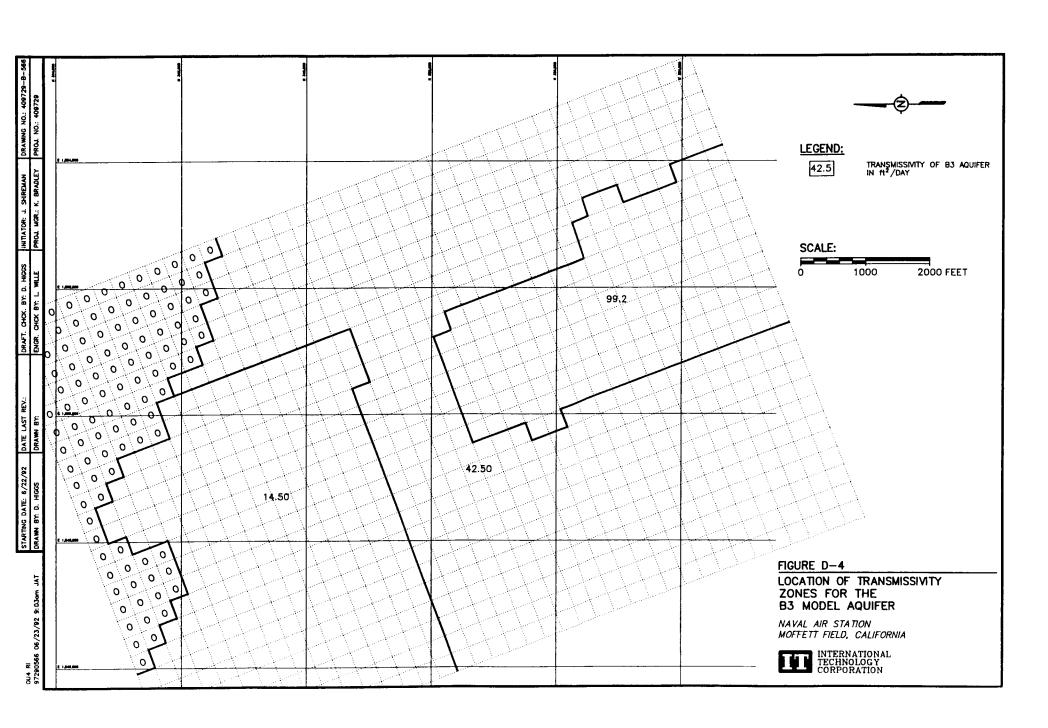


Table D.1-5

Transmissivity Values Assigned to Model Zones for B2- and B3-Aquifers

	Thickness Zones					
	b = 5	b = 10	b = 15	b = 25	b = 35	b = 40
Hydraulic Conductivity	T ft <sup>2</sup> /day	T ft <sup>2</sup> day				
For B2 and B3 K = 2.83 ft/day	14.3	28.3	42.5	70.9	99.2	113.0

# **D.1.2 Boundary Conditions**

The major recharge areas for the A1-, A2-, B2-, and B3-aquifer zones (Moffett designation) are believed to be outside of the Moffett Field and MEW areas (PRC, 1991); therefore, these recharge areas are treated as general head boundaries in the model. Heads for the boundaries at Column 39 were projected from the water table surface maps for May 1991 for aquifers A1 and A2, (Figures L-1 and L-5, IT, 1991d). Therefore, the head values at the southern boundaries are dependent on and proportional to heads outside of the model area. Head values for the northern boundaries are controlled by heads imposed by the bay and water withdrawal at the drainage sump and are assumed to be consistent head boundaries for this model.

The values for the general head boundaries were derived from water elevation contours as shown in the above referenced figures. The southern boundary values appear to be approximately 2 feet lower in the A2-aquifer than in the A1-aquifer.

#### D.1.3 Leakance Terms

Hydraulic conductivities were derived from test analysis of aquifers based on evaluation of data and an assumed leakance factor (computed by the model from values of vertical hydraulic conductivity and estimated aquitard thickness, which are input terms) and assuming 100 percent contribution of inflow from either the overlying or underlying aquitard (HLA, 1988). (Vertical conductivities for the A1/A2, A2/B2, and B2/B3 aquitards are reviewed in IT calculation sheets, 1991c.) Table D.1-6 indicates the order of magnitude of the hydraulic conductivity in the vertical direction (K<sub>v</sub>).

Table D.1-6

Input Values for Calculation of Leakance Term for Aquitard Layers

Aquitard	K <sub>v</sub> (cm/s) <sup>a</sup>	
A1/A2	1 x 10 <sup>-5</sup>	
A2/B2	1 x 10 <sup>-6</sup>	
B2/B3	1 x 10 <sup>-4</sup>	

 $<sup>{}^{</sup>a}K_{v}$  = hydraulic conductivity in the vertical direction.

Leakance terms required by MODFLOW (McDonald and Harbaugh, 1988) are defined as K<sub>v</sub>/b where b is the aquitard thickness. Aquitard thickness was estimated from the depth to the bottom and top of the respective aquifers. Vertical hydraulic conductivities for the A1/A2 and A2/B2 aquitards are given in Table D.1-6.

Aquitard thickness for each model layer was estimated from the elevations of the base of one aquifer and the top of the underlying aquifer as shown in the structural contour maps provided in IT calculation check sheets (IT, 1991a).

#### D.1.4 Calibration

The initial model runs for each aquifer layer did not produce satisfactory reproduction of steady-state water elevations at the facility. Specifically, the position of the sea level contour line was shifted too far north, and the curvature of the contour lines did not match the contour shapes as shown in IT 1991b. Although some uncertainty in contour curvature and placement exists because of the limited number and areal distribution of control points, the initial simulation was not close to site conditions. Therefore, the position and order of magnitude of the aquifer parameter zones were adjusted to produce a more acceptable water table configuration. This adjustment was justifiable because more is known about the water table surface than about the position of changes in material properties of the aquifers.

Hydraulic conductivities for the east side of the facility were adjusted downward in the area of Sites 5, 6, and 7, which caused displacement of the zero lines in the A1- and A2-aquifer zones to the south, more in line with observed site conditions.

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Modeled drains were located in Layer 1 only (A1-aquifer zone). The model defines a drain as a sink when groundwater elevation is above a predetermined elevation within the node. If the groundwater elevation is below the predetermined elevation, the node will be inactive. The effect of the drain in Layer 1 was very evident in Layer 2, matching the effects seen in the observed data. This demonstrates leakance between the A1- and A2-aquifer zones at this location.

Transmissivity was reduced in the area of Sites 5, 6, and 7 for the B2-aquifer so that water levels would more closely match the observed data.

While calibration was based on comparison of the water table configuration, model water levels were numerically compared to the average water elevations collected during the Second, Third, and Fourth Quarters of 1990 and the First Quarter of 1991. The final comparisons are given in Table 5.2-3 (Chapter 5.0). Monitoring wells were located by the grid node in which they fell. Where a well was on or near a block boundary, model levels in the adjoining grid blocks were averaged for comparison. Actual water levels used for comparison are averages of up to four quarterly water level measurements from 1990 to 1991. The mean of the difference between average observed and model water levels for the A1-aquifer is 0.16 foot (standard deviation [SDEV] = 2.16 feet), for the A2-aquifer 0.95 foot (SDEV = 1.95), and for the B2-aquifer 2.15 feet (SDEV = 1.57). There is only one data point for the B3-aquifer, and the predicted head is 2.59 feet higher than the historical average head in that well. Comparisons should not be used to statistically evaluate the model results with respect to individual wells.

Modeling of the groundwater levels for the B2-aquifer closely matches actual conditions of that aquifer at the base. Transmissivity domains were adjusted slightly, and hydraulic conductivities were reestimated during calibration. It was found that the adjustments made did not markedly affect the absolute magnitude or gradient of heads within the B2-aquifer; however, some changes were noted in the hydraulic head levels in both the A1- and A2-aquifers.

Similarly, comparison of actual water levels (average of all data) and modeled water levels did not compare favorably when comparisons were made to the initial runs for the A1, A2, and B2-aquifers. Hydraulic conductivity domains and transmissivity domains were shifted or multiplied by factors of 10 to establish the sensitivity of the model to these parameters. It was found that the model was not greatly sensitive to changes in transmissivity within the

confined layers but did appear to be somewhat more sensitive to changes in hydraulic conductivity in the uppermost unconfined layer.

### D.1.5 References for Flow Model Development

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Freeze, L. A. and V. A. Cherry, 1979, *Groundwater*, Prentice-Hall Inc., Englewood Cliffs, New Jersey.

Harding Lawson Associates (HLA), 1987, Remedial Investigation, Middlefield-Ellis-Whisman Area, Mountain View, California.

Harding Lawson Associates (HLA), 1988, Remedial Investigation Report; RI/FS Middlefield-Ellis-Whisman Area, Mountain View, California.

IT, 1991a, Thickness of Aquifer Units at NAS Moffett Field, IT Calculations and Calculation Check Prints.

IT, 1991b, Site Characterization Report, NAS, Moffett Field.

IT, 1991c, Hydraulic Conductivities for Aquifers A to B3 MEW Sites Based on Results of Harding Lawson Pump Tests, IT Calculation Check Prints.

IT, 1991d, Quarterly Status Report for the 3rd Quarter 1991, Moffett Naval Air Station.

McDonald and Harbaugh, 1988, A Modular Three Dimensional Finite Difference Ground-water Flow Model, U.S. Geologic Survey Techniques of Water Resources Investigations, Book 6.

PRC, 1991, Draft Operable Unit 4 Technology Screening Report, CLEAN Contract No. N62474-88-D5086.

# D.2.0 Solute Transport Model

# D.2.1 Transport Model Development

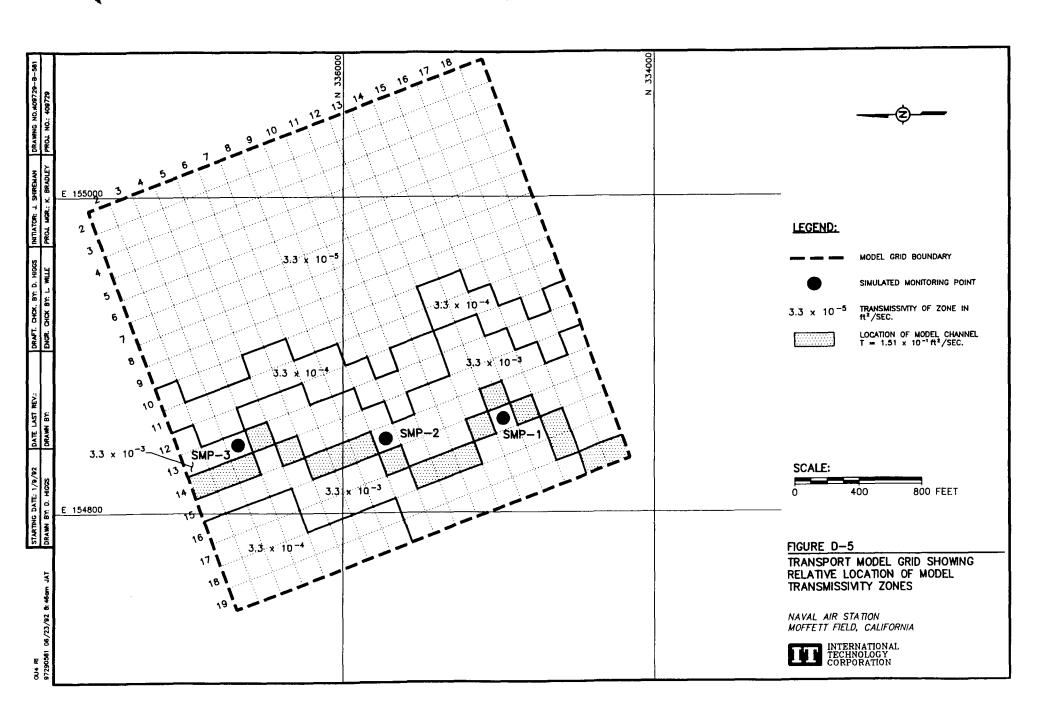
The solute transport model, MOC, used for the west side aquifers is a two-dimensional finite difference model developed by the U.S. Geological Survey (USGS). The A1- and A2-aquifer zones exhibit the most significant impact by chlorinated solvents, which form the most extensive plume at Moffett Field. Therefore, these aquifers were chosen for solute transport modeling.

Figure 3.6-1 of this RI report provides comparisons between aquifer zones at Moffett Field, the MEW area, and terminology used by PRC (1991). Thus, for the modeling of solute transport, one layer can represent the A1- and A2-aquifer zones. Aquifer testing at Moffett Field has shown that the A1- and A2-aquifer zones are hydrogeologically connected (see Appendix F). The objectives of the modeling were (1) to establish whether the observed plume configuration could be due to encroachment of a plume front migrating into the Moffett Field area from off site and (2) to estimate the magnitude of on-site sources, if required, that would account for observed concentrations that cannot be accounted for based on off-site sources only.

The model area was telescoped down to the Site 9 area because this area of the west side aquifer exhibits the most significant impact of groundwater by a suite of organic compounds, most notably trichloroethene (TCE) and tetrachloroethylene (PCE). (Groundwater at Sites 8 and 12 exhibits less impact by organic compounds.)

In the flow modeling (see Chapter D.1.0) at Moffett Field, the observed configuration of groundwater levels was closely matched by utilizing a relatively heterogeneous hydraulic conductivity field or transmissivity field. For contaminant transport modeling, the same values were applied. However, because a much smaller region was modeled, more detail could be incorporated into the aquifer model, such as sandy deposits near gravel filled channels. Transmissivities for the modeled aquifer zones are shown in Figure D-5.

Geochemical data required for the transport model were derived from information available in the Superfund Public Health Manual (U.S. EPA, 1986) and from the literature. The main geochemical datum required by the model is the distribution coefficient  $(K_d)$ .  $K_d$  is computed as a function of the organic carbon partition coefficient  $(K_{oc})$ , the organic carbon fractions of



the compounds of the medium, and fraction of silt and sand. The method used to compute  $K_d$  follows the method outline in U.S. EPA, 1987 (page 51). From U.S. EPA 1986,  $K_{oc}$  for TCE is 126 mL/kg. From Roberts, et al. (1991), the fraction of organic carbon in silts is 0.004 mg/kg and 0.0007 mg/kg for sands. The distribution of silt and sand in the A1- and A2-aquifer zones is assumed to be 0.20 silt and 0.80 sand (Table D.2-1).

The most important assumption concerning the solute transport is that transport is controlled by a linear adsorption reaction. The mathematical treatment is given in Konikow and Bredehoeft (1978) and Goode and Konikow (1989).

Known concentrations in groundwater at suspected upgradient sources were used to develop input data for model contaminant sources. Two contaminant source types were modeled: a boundary source that simulated encroachment of an external plume to Site 9 and an injection source to model on-site sources. Review of quarterly data suggests that concentrations within the MEW area have not changed significantly and that this area can act as a general source. The modeling has been used to estimate the mode of development of the VOC plume. TCE was chosen as a representative component for indicating VOC because of its high concentration in the upgradient area and its low organic carbon distribution coefficient.

Table D.2-1 Input Values used in the Transport Modeling

I. Finite Difference Grid	10.37	150.07
X Axis (active nodes)	18 Nodes	150 ft/node
Y Axis (active nodes)	18 Nodes	150 ft/node
II. Hydrogeologic		
Parameters		
Material Type	<u>K (cm/s)</u>	Thickness
Silty sand (zone)	1 x 10 <sup>-4</sup>	10 ft
Fine sand	1 x 10 <sup>-3</sup>	10 ft
Coarse sand	1 x 10 <sup>-1</sup>	10 ft
Sandy gravel	3.1 x 10 <sup>-1</sup>	15 ft
Precipitation		Pavement prevents recharge
Flow regime	Steady state	
Boundary Conditions		Constant head
North boundary		2 to 9 ft msl <sup>a</sup>
South boundary		20 to 23 ft msl
		20 to 25 it inst
III. Geochemical		
Parameters		
Source Type	Concentration	Rate
Constant Head 2	3,700 ppb	
Constant Head 3	6,700 ppb	0.000 ch
Injection	10,000 ppb	0.002 cfs <sup>b</sup>
Distribution coefficient	0.17 mL/g <sup>c</sup>	
Bulk density	1.8 g/mL <sup>d</sup>	
$X_{oc}^{f}$	0.004 (Roberts, et al., 1990)	
X <sub>oc</sub> f	0.0007 (Poborts et al	
	0.0007 (Roberts, et al., 1990)	
	1990)	

<sup>\*</sup>msl - mean sea level.

<sup>&</sup>lt;sup>b</sup>cfs - cubic feet per square inch.

cmL/g - milliliter per gram.
dg/mL - gram per milliliter.

 $<sup>{}^{</sup>c}X_{oc}^{f}$  Fraction of organic carbon in the fine grain material.

 $<sup>{}^{</sup>r}X_{oc}^{s}$  Fraction of organic carbon in the sand material.

# D.2.2 References for Solute Transport Modeling

Goode, D. J. and L. F. Konikow, 1989, Modifications of a Method-of-Characteristics Solute-Transport Model to Incorporate Decay and Equilibrium Controlled Sorption or Ion Exchange, U.S. Geologic Survey, Water Resources Investigations Report 89-4080.

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IT, 1991b, Site Characterization Report for Moffett Naval Air Station.

Konikow, L. F. and J. D. Bredehoeft, 1978, Techniques of Water-Resources Investigations, Computer Model of Two Dimensional Waste Transport and Dispersion in Groundwater, U.S. Geologic Survey, Water Resources Division.

PRC Environmental Management, Inc., 1991, "Building 29 Area Field Investigation Technical Memorandum," CLEAN Contract No. N62474-88-D5086.

Roberts, P. V., G. D. Hopkins, D. M. Mackay, and L. Semrini, 1990, "A Field Evaluation of In-Situ Biodegradation of Chlorinated Ethenes: Part I, Methodology and Field Site Characterization," *Ground Water*, Vol. 28, No. 4.

U.S. EPA, 1986, Superfund Public Health Evaluation Manual, Environmental Protection Agency, U.S. EPA/540/1-86/060.

U.S. EPA, 1985, Water Quality Assessment - A Screening Procedure for Toxic and Conventional Pollutants in Surface and Groundwater Part II, Environmental Protection Agency U.S. EPA/600/6-85/0026.

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# APPENDIX D – GROUNDWATER FLOW MODEL AND SOLUTE TRANSPORT MODEL

THIS RECORD CONTAINS LARGE VOLUMES OF DATA AND IS NOT REQUIRED TO BE PHYSICALLY LOCATED WITH THE ADMINISTRATIVE RECORD DOCUMENT.

DUE TO EXTENSIVE VOLUME, THIS DATA WILL NOT BE IMAGED.

TO VIEW THE DATA, CONTACT:

DIANE C. SILVA
RECORDS MANAGEMENT SPECIALIST
NAVAL FACILITIES ENGINEERING COMMAND
SOUTHWEST
1220 PACIFIC HIGHWAY
SAN DIEGO, CA 92132

**TELEPHONE:** (619) 532-3676

# VOLUME 4, APPENDIX E RISK ASSESSMENT MODELS

# Appendix E

# E.1.0 Modeling

## **Indoor Air Modeling**

Models used to describe potential indoor concentrations are equations describing use of groundwater as potable water. These models are volatilization while showering (Murphy, 1987) and from general household water use (Murphy, 1987). Henry's Law is used to describe partitioning from the water to the air; hence, volatilization is directly proportional to the magnitude of the Henry's Law Constant. The general water use model describes volatilization from total water use. This model assumes that all water use occurs at a single average temperature, and thus does not precisely account for individual uses such as cooking.

#### Volatilization while showering

$$D_s = 10^3 (FC/V_a)[1+1/aT_s(e^{-aT_s}-1)][1-exp-[0.93+(1.48 \times 10^{-3}/H^{-1})]$$

where:

 $C_s$  = concentration while showering (mg/m<sup>3</sup>)

 $F = \text{shower water flow rate } (0.48 \text{ m}^3/\text{hr})$ 

C = concentration of chemical in water (chemical-specific, mg/L)

V = volume of shower/bathroom (12 m<sup>3</sup>)

a = air exchange rate between shower/bathroom and rest of house (12 hr<sup>-1</sup>)

H = Henry's Law Constant (chemical-specific, m<sup>3</sup>-atm/mole)

# Volatilization from general water use

$$C_h = (Q_w/Q_a)MC[1-exp-[1.26 + (2.00 \times 10^{-3})^{-1}]$$

where:

C<sub>h</sub> = concentration from general household water use (mg/m<sup>3</sup>)

 $Q_w = \text{water use in home (980 l/day)}$ 

 $Q_a$  = volume of air exchange rate for home (8,700 m<sup>3</sup>/day)

M = mixing factor (0.5 unitless)

#### Concentration in Vegetables

The following model (modified from NRC, 1977) was used to estimate potential concentrations of chemicals in leafy vegetables due to deposition of irrigation water onto the soil and the exposed portions of plants:

$$C_{v} = C_{v} I_{r} \left[ \frac{F_{r}(1 - e^{ht})}{(K*Y)} + \frac{F_{r} B_{i} T_{s}}{D_{s}} \right]$$

where:

 $C_v = \text{concentration in vegetables (mg/kg)}$ 

 $C_w = \text{concentration in water (mg/L)}$ 

I<sub>r</sub> = annual irrigation rate = 0.097 1/m<sup>2</sup>/hr (Baes et al., 1984)

 $F_{r}$  = fraction of irrigation water retained on plant surface (unitless) = 0.25 (NRC, 1977)

K = removal rate constant from weathering = 0.0021 hr<sup>-1</sup>

t = length of time plant is exposed = 1,440 hr (NRC, 1977)

Y = agricultural productivity yield = 1.0 kg/m<sup>2</sup> (Baes et al., 1984)

 $F_i$  = fraction of year that irrigation occurs (unitless) = 0.58 (Baes et al., 1984)

B<sub>i</sub> = chemical-specific root uptake factor -- transfer to vegetative portion of plant

 $T_s$  = time soil is exposed to irrigation = 131,000 hr (NRC, 1977)

D<sub>e</sub> = effective soil surface density =  $240 \text{ kg/m}^2$  (NRC, 1977)

It was assumed that leafy vegetables would be grown as these will intercept the greatest amount of irrigation water on the edible plant surface. Contamination may result through both direct deposition of irrigation water onto the edible portion of the plant and uptake of the water by the roots from the soil. It was assumed that vegetables will be eaten raw and unwashed, thus eliminating these potential removal mechanisms.

# E.2.0 Concentration/Toxicity Screen for Chemicals of Potential Concern

Infrequently detected chemicals (<5 percent frequency of detection) were excluded as chemicals of potential concern. However, if a chemical was analyzed for in less than 20 samples at a site, a single positive hit would result in greater than 5 percent detection; therefore, chemicals with fewer than 20 samples analyzed were evaluated on a case-by-case basis. The chemicals' toxicity and concentration were considered in this evaluation.

Chemicals were evaluated by estimating a potential ILCR and/or HQ for a worst-case residential exposure to the maximum concentration of that chemical at the site. Intakes were estimated for estimated for drinking water ingestion by adults using the equations shown in Section 6.3.3.1. The exposure parameters are shown below:

Parameter	Value	Reference
Drinking water ingestion rate	2 L/day	U.S. EPA, 1991a
Exposure frequency	365 days/yr	Worst-case assumption
Exposure duration	70 years	Worst-case assumption
Body weight	70 kg	U.S. EPA, 1991a
Averaging time	25,550 days	U.S. EPA, 1989a

Four chemicals were excluded because they were detected only once at a site and had very low toxicity at the detected concentration. The results of the toxicity screen for these chemicals is shown below:

Chemical	Exposure	Estimated		
	Concentration <sup>a</sup> (mg/L)	Intake (mg/kg/day)	CPF (mg/kg/day)	Estimated ILCR
Trichloroethene	0.002	5.7 x 10 <sup>-5</sup>	1.1 x 10 <sup>-2</sup>	6.3 x 10 <sup>-7</sup>

Chemical	Exposure Concentration <sup>a</sup> (mg/L)	Estimated Intake (mg/kg/day)	RfD (mg/kg/day)	Estimated HQ
Benzoic acid	0.025	6.0 x 10 <sup>-10</sup>	4.0 x 10°	1.5 x 10 <sup>-10</sup>
2-Butanone	0.15	$4.3 \times 10^{-3}$	5.0 x 10 <sup>-2</sup>	8.6 x 10 <sup>-2</sup>
Nickel	40	9.6 x 10 <sup>-7</sup>	2.0 x 10 <sup>-2</sup>	4.8 x 10 <sup>-5</sup>

# E.3.0 Hazard Identification

The information in this section comes from IRIS (U.S. EPA, 1992).

**Acetone.** An RfD of 1 x 10<sup>-1</sup> mg/kg/day, an NOAEL of 100 ng/kg/day, and an LOAEL of 500 mg/kg/day were reported with an uncertainty factor of 1000, 100 for inter- and intraspecies extrapolation and 10 to extrapolate from subchronic to chronic exposure.

Acetone was administered by gavage to groups of albino rats at three different levels. Body weights, food consumption, clinical chemistry, hematology, and histopathologic parameters, as well as organ weights and organ-to-body weight ratios, were measured and analyzed. Animals were sacrificed after 30 or 90 days of exposure. No effects were seen at the low dose level throughout the study. RBC parameters were significantly increased in the high group at 30 days (males only) and at 90 days in males and females. Statistical analysis of the absolute and relative organ weight data revealed significantly increased kidney weights for females in the medium and high dosage groups and increased kidney-to-body and brain weight ratios for males and females in the high doses. Liver weight and liver/body weight ratios were also increased in the high dose males and females. Histopathologic studies revealed a marked increase in severity in tubular degeneration of the kidneys and hyaline droplet accumulation with increasing doses. This accumulation was significant in the medium and high males and the high females.

Based on the above findings, the NOEL for this study is 100 mg/kg/day and the LOAEL is 500 mg/kg/day based on increased liver and kidney weights and nephrotoxicity.

Limited human studies have shown that workers exposed to acetone vapors experienced transient eye and nose irritation. Animals exposed to acetone vapors experienced slight, but not significant, decreases in organ and body weights.

Confidence in the principal study is rated medium, since a moderate number of animals/dose/sex and an extensive number of parameters were measured. The data base is rated low because a very limited number of studies are available and no pertinent supporting studies were located. The overall confidence rating for the RfD is low.

Acetone is classified as a class D carcinogen based on lack of data concerning carcinogenicity in humans or animals.

Acetone did not show mutagenic activity when tested in Salmonella typhimurium strains TA98 and TA100 or in Schizosaccharomyces pombe strain P1 either in the presence or absence of liver homogenates or in cell transformation systems. Furthermore, acetone gave negative results in assays for chromosomal aberrations and sister chromatid exchange, DNA binding, point mutation in mouse lymphoma cells, and transfection of E. coli CR63 cells. In one study, however, acetone was reported to produce chromosomal aberrations but not sister chromatid exchanges.

Arsenic. An RfD of 3 x 10<sup>-4</sup> mg/kg/day, an NOAEL of 0.008 mg/kg/day and an LOAEL of 0.014 mg/kg/day were reported with an uncertainty factor of 3. The UF of 3 is to account for both the lack of data to preclude reproductive toxicity as a critical effect and to account for some uncertainty in whether the NOAEL of the critical study accounts for all sensitive individuals. The data reported in Tseng show an increased incidence of blackfoot disease that increases with age and dose. Blackfoot disease is a significant adverse effect. The prevalences (males and females combined) at the low dose are 4.6 per 1000 for the 20-39 year group, 10.5 per 1000 for the 40-59 year group, and 20.3 per 1000 for the >60 year group. Moreover, the prevalence of blackfoot disease in each age group increases with increasing dose. However, a recent report indicates that it may not be strictly due to arsenic exposure. The data in Tseng also show increased incidences of hyperpigmentation and keratosis with age. The overall prevalences of hyperpigmentation and keratosis in the exposed groups are 184 and 71 per 1000, respectively. The text states that the incidence increases with dose, but data for the individual doses are not shown. These data show that the skin lesions are the more sensitive endpoint. The low dose in the Tseng study is considered a LOAEL.

Ferm and Carpenter produced malformations in 15-day hamster fetuses via intravenous injections of sodium arsenate into pregnant dams. Exencephaly, encephaloceles, skeletal defects and genitourinary systems defects were produced. These and other terata were produced in mice and rats at similar exposure levels. Minimal effects or no effects on fetal development have been observed in studies on chronic oral exposure of pregnant rats or mice to relatively low levels of arsenic via drinking water. Nadeenko reported that intubation of rats with arsenic solution during pregnancy, produced no significant embryotoxic effects and only infrequent slight expansion of ventricles of the cerebrum, renal pelves and urinary bladder. Hood reported that very high single oral doses of arsenate solutions to pregnant mice were necessary to cause prenatal fetal toxicity, while multiple doses of lower concentrations had little effect.

Extensive human pharmacokinetic, metabolic, enzymic and long-term information is known about arsenic and its metabolism. Valentine established that human blood arsenic levels did not increase until daily water ingestion of arsenic exceeded approximately 250 ug/day. Methylated species of arsenic are successively 1 order of magnitude less toxic and less teratogenic. Some evidence suggests that inorganic arsenic is an essential nutrient in goats, chicks, mini pigs and rats. No comparable data are available for humans. Confidence in the chosen study is considered medium. An extremely large number of people were included in the assessment (>40,000) but the doses were not well-characterized and other contaminants were present. The supporting human toxicity data base is extensive but somewhat flawed. Problems exist with all of the epidemiological studies.

Arsenic is classified as a class A carcinogen based on observation of increased lung cancer mortality in populations exposed primarily through inhalation and on increased skin cancer incidence in several populations consuming drinking water with high arsenic concentrations.

Studies of smelter worker populations (Tacoma, WA; Magma, UT; Anaconda, MT; Ronnskar, Sweden; Saganoseki-Machii, Japan) have all found an association between occupational arsenic exposure and lung cancer mortality. Both proportionate mortality and cohort studies of pesticide manufacturing workers have shown an excess of lung cancer deaths among exposed persons. One study of a population residing near a pesticide manufacturing plant revealed that these residents were also at an excess risk of lung cancer. Case reports of arsenical pesticide applicators have also demonstrated an association between arsenic exposure and lung cancer.

A cross-sectional study of 40,000 Taiwanese exposed to arsenic in drinking water found significant excess skin cancer prevalence by comparison to 7500 residents of Taiwan and Matsu who consumed relatively arsenic-free water. This study design limited its usefulness in risk estimation. Arsenic-induced skin cancer has also been attributed to water supplies in Chile, Argentina and Mexico. No excess skin cancer incidence has been observed in U.S. residents consuming relatively high levels of arsenic in drinking water. The results of these U.S. studies, however, are not necessarily inconsistent with the existing findings from the foreign populations. The statistical powers of the U.S. studies are considered to be inadequate because of the small sample size.

A follow-up study of the population living in the same area of Taiwan, where arsenic contamination of the water supply was endemic, found significantly elevated standard

mortality ratios for cancer of the bladder, lung, liver, kidney, skin and colon. This study of bladder, liver and lung cancer cases in the endemic area found a significant association with arsenic exposure that was dose-related. The association of arsenic ingestion and cancer of various internal organs has also been cited in a number of case reports. Persons treated with arsenic-containing medicinals have also been shown to be at a risk of skin cancer.

There has not been consistent demonstration of arsenic carcinogenicity in test animals for various chemical forms administered by different routes to several species. There are some data to indicate that arsenic may produce animal tumors if retention time in the lung can be increased.

Sodium arsenate has been shown to transform Syrian hamster embryo cells and to produce sister-chromatid-exchange in DON cells, CHO cells and human peripheral lymphocytes exposed in vitro. While arsenic compounds have not been shown to mutate bacterial strains, it produces preferential killing of repair deficient strains.

**Barium.** An RfD of  $7 \times 10^{-2}$  mg/kg/day and an NOAEL of 0.21 mg/kg/day were reported with an uncertainty factor of 3. Because of both the critical study's unique focus and the supporting studies, a 3-fold UF, instead of a 10-fold UF, was chosen as most appropriate to protect for sensitive individuals within that population.

Wones administered barium (as barium chloride) in the drinking water of 11 healthy male volunteers. There were no changes in systolic or diastolic blood pressures, orserum chemistry, especially total cholesterol, HDL, LDL, triglycerides, potassium or glucose levels. There was an increase in serum calcium levels that was attributed to a decrease in serum albumin levels. This increase, although statistically significant, was considered borderline and not clinically significant. There were also no changes in cardiac cycle as noted by electrocardiograms and no significant arrhythmias. A NOAEL of 10 mg/L was identified in this study which corresponds to 0.21 mg/kg/day, based on an actual consumption rate of 1.5 L/day and a 70-kg body weight.

Occupational studies of workers exposed to barium dust have shown that workers develop "baritosis." Affected workers showed no symptoms, no abnormal physical signs, no loss of vital capacity or interference with function, although they had a significantly higher incidence of hypertension.

McCauley studied the histologic and cardiovascular effects of drinking water containing barium on Sprague-Dawley rats. No significant histologic, carcinogenic, or cardiovascular (including hypertension) effects were observed. No changes were reported in body weight, or food and water consumption in any of the treated animals. Animals treated at the highest dose did exhibit ultrastructural changes in the kidney glomeruli and the presence of myelin figures. No other effects were reported at any dose level for males or females.

Perry exposed weanling rats to barium. There were no signs of toxicity at any barium dose level. Systolic blood pressure measurements revealed no increase in animals.

EPA does not believe that any single study, considered alone, is adequate to calculate an RfD for barium. However, EPA believes that medium confidence can be placed in the total data base used to determine the RfD.

A risk assessment for this substance/agent is under review by an EPA work group.

**Benzene.** No reference dose is available for benzene. A risk assessment for this substance/agent will be reviewed by an EPA work group.

Benzene is classified as a group A carcinogen. This classification is based on several studies of increased incidence of nonlymphocytic leukemia from occupational exposure, increased incidence of neoplasia in rats and mice exposed by inhalation and gavage, and some supporting data.

Aksoy reported effects of benzene exposure among 28,500 Turkish workers employed in the shoe industry. Mean duration of employment was 9.7 years (1-15 year range) and mean age was 34.2 years. Peak exposure was reported to be 210-650 ppm. Twenty-six cases of leukemia and a total of 34 leukemias or preleukemias were observed, corresponding to an incidence of 13/100,000 (by comparison to 6/100,000 for the general population). A follow-up paper (Aksoy, 1980) reported eight additional cases of leukemia as well as evidence suggestive of increases in other malignancies.

In a retrospective cohort mortality study Infante examined leukemogenic effects of benzene exposure in 748 white males exposed while employed in the manufacturing of rubber products. A statistically significant increase (p less than or equal to 0.002) of leukemias was

found by comparison to the general U.S. population. There was no evidence of solvent exposure other than benzene.

In a subsequent retrospective cohort mortality study Rinsky observed seven deaths from leukemia among 748 workers exposed to benzene and followed for at least 24 years (17,020 person-years). This increased incidence was statistically significant.

In an updated version of the Rinsky study, the authors followed the same cohort to 12/31/81. An in his earlier study, cumulative exposure was derived from historic air-sampling data or interpolated estimates based on exisiting data. Standardized mortality rates ranged from 109 at cumulative benzene exposures under 40 ppm-years and increased montonically to 6637 (6 cases) at 400 ppm-years or more. The authors found significantly elevated risks of leukemia at cumulative exposures less than the equivalent current standard for occupational exposure which is 10 ppm over a 40-year working lifetime.

Ott observed three deaths from leukemia among 594 workers followed for at least 23 years in a retrospective cohort mortality study, but the increase was not statistically significant. Exposures ranged from <2 to >25 ppm 8-hour TWA.

Wong reported on the mortality of male chemical workers who had been exposed to benzene for at least 6 months during the years 1946-1975. The study population of 4062 persons was drawn from seven chemical plants. Dose-dependent increases were seen in leukemia and lymphatic and hematopoietic cancer. The incidence of leukemia was responsible for the majority of the increase. It was noted that the significance of the increase is due largely to a less than expected incidence of neoplasia in the unexposed subjects.

Numerous other epidemiologic and case studies have reported an increased incidence or a causal relationship between leukemia and exposure to benzene. In addition to this human data, both gavage and inhalation exposure of rodents to benzene have resulted in development of neoplasia. Numerous investigators have found significant increases in chromosomal aberrations of bone marrow cells and peripheral lymphocytes from workers with exposure to benzene. Benzene also induced chromosomal aberrations in bone marrow cells from rabbits, mice and rats. Several investigators have reported positive results for benzene in mouse micronucleus assays. Benzene was not mutagenic in several bacterial and yeast systems, in the sex-linked recessive lethal mutation assay with Drosophila melanogaster or in mouse lymphoma cell forward mutation assay.

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Based on these studies the EPA recommends an inhalation unit risk of 8.3E-6 per (ug/cu.m) using the One-hit (pooled data) dose extrapolation method. This unit risk is based on occupational studies on humans which showed increased incidence of leukemia. The unit risk estimate is the geometric mean of four ML point estimates using pooled data from the Rinsky and Ott studies, which was then adjusted for the results of the Wong study. The Rinsky data used were from an updated tape which reports one more case of leukemia than was published in 1981. Equal weight was given to cumulative dose and weighted cumulative dose exposure categories as well as to relative and absolute risk model forms. The results of the Wong study were incorporated by assuming that the ratio of the Rinsky-Ott-Wong studies to the Rinsky-Ott studies for the relative risk cumulative dose model was the same as for other model-exposure category combinations and multiplying this ratio by the Rinsky-Ott geometric mean. The age-specific U.S. death rates for 1978 (the most current year available) were used for background leukemia and total death rates. It should be noted that a recently published paper reported yet another case of leukemia from the study population.

An oral slope factor of 2.9E-2 per (mg/kg)/day is recommended based on human data for inhalation exposure. The human respiratory rate was assumed to be 20 cu.m/day and the human drinking water intake was assumed to be 2 L/day. The fraction of the administered dose absorbed systemically via inhalation and via drinking water were assumed to be equal.

**Beryllium.** An oral RfD of  $5 \times 10^{-3}$  mg/kg/day and an NOAEL of 0.54 mg/kg/day were reported with an uncertainty factor of 100. The uncertainty factor of 100 reflects a factor of 10 each for interspecies conversion and for the protection of sensitive human subpopulations.

Fifty-two weanling Long-Evans rats of each sex received beryllium (as BeSO4, beryllium sulfate) in drinking water. Exposure was for the lifetime of the animals. At natural death the rats were dissected and gross and microscopic changes were noted in heart, kidney, liver, and spleen. There were no effects of treatment on these organs or on lifespan, urinalysis, serum glucose, cholesterol, and uric acid, or on numbers of tumors. Male rats experienced decreased growth rates from 2 to 6 months of age.

Similar studies were carried out on Swiss (CD strain) mice. Female animals showed decreased body weight compared with untreated mice. Male mice exhibited slight increases in body weight. These effects were not considered adverse, therefore, 0.95 mg/kg/day is considered a NOAEL.

This RfD is limited to soluble beryllium salts. Data on the teratogenicity or reproductive effects of beryllium are limited. It has been reported to produce embryolethality and terata in chick embryos.

Confidence in the study is rated as low because only one dose level was administered. Although numerous inhalation investigations and a supporting chronic oral bioassay in mice exist, along with the work by Cox which indicates that a higher dose level might be a NOEL, these studies are considered as low to medium quality; thus, the data base is given a low confidence rating. The overall confidence in the RfD is low, reflecting the need for more toxicity data by the oral route.

The 1985 Drinking Water Criteria Document for Beryllium is currently undergoing Agency review.

Beryllium is considered a B2 probable human carcinogen. Beryllium has been shown to induce lung cancer via inhalation in rats and monkeys and to induce osteosarcomas in rabbits via intravenous or intramedullary injection. Human epidemiology studies are considered to be inadequate.

Human carcinogenicity was considered inadequate. Reported increases, while apparently associated with exposure, did not take a variety of possible confounding factors into account.

Wagoner observed 47 deaths from cancer among 3055 white males employed in beryllium-processing with a median duration of employment of 7.2 months. Among the 2068 followed for 25 years or more, 20 lung cancer deaths were observed. These increased incidences were statistically significant. When lung cancer mortality data became available for 1968-1975, the number of expected deaths was recalculated and the increased incidence was statistically significant only among workers followed 25 years or more. When the number of expected deaths was adjusted for smoking, the increased incidence was no longer significant.

An earlier study of workers from this same beryllium processing plant, and several studies of workers from this plant combined with workers from other beryllium plants, have reported a statistically significant increased incidence of lung cancer. No adjustment was made for smoking in these studies, and all were limited in their ability to detect a possible increased incidence of lung cancer because of methodological constraints and deficiencies.

Animal carcinogenicity is adequate and based on the evidence for induction of tumors by a variety of beryllium compounds in male and female monkeys and in several strains of rats of both sexes, via inhalation and intratracheal instillation, and the induction of osteosarcomas in rabbits by intravenous or intramedullary injection in multiple studies.

Slight increases in cancer incidence (not statistically significant in comparison with controls) were reported in Long-Evans rats administered 5 ppm beryllium sulfate in the drinking water for a lifetime. The authors reported a slight excess of grossly observed tumors in the 5 ppm group over controls in the male rats. The power of this test to detect a carcinogenic effect was reduced by high mortality. Schroeder and Mitchener administered 5 ppm beryllium sulfate in drinking water to Swiss mice over a lifetime. A non-statistically significant increase in incidence of lymphoma leukemias were reported in the females relative to controls.

An increase in reticulum cell sarcomas of the lungs was seen in male, but not female Wistar-derived rats administered beryllium sulfate in the diet.

Osteogenic sarcomas were induced in rabbits by intravenous injection of beryllium compounds in at least 12 different studies and by intramedullary injection in at least four studies. Bone tumors were induced by beryllium oxide, zinc beryllium silicate, beryllium phosphate, beryllium silicate and beryllium metal. No bone tumors were reported to be induced by intravenous injection of beryllium oxide or zinc beryllium silicate in rats or guinea pigs. Positive results, however, were reported in mice injected with zinc beryllium silicate, although the numbers were not listed. The sarcomas were generally reported to be quite malignant and metastasized to other organs.

Lung tumors, primarily adenomas and adenocarcinomas, have been induced via the inhalation route in both male and female Sprague-Dawley rats during exposure periods of up to 72 weeks by beryllium sulfate, in both male and female Sherman and Wistar rats by beryllium phosphate, beryllium fluoride and zinc beryllium silicate, in male Charles River CR-CD rats by beryl ore and in both male and female rhesus monkeys by beryllium sulfate. Positive results were seen in rats exposed to beryllium sulfate at concentrations as low as 2 ug/cu.m.

Tumors were also induced by intratracheal instillation of metallic beryllium, beryllium-aluminum alloys and beryllium oxide in both Wistar rats and rhesus monkeys.

Adenomas, adenocarcinomas and malignant lymphomas were seen in the lungs, with lymphosarcomas and fibrosarcomas present at extrapulmonary sites.

Beryllium sulfate and beryllium chloride have been shown to be nonmutagenic in bacterial and yeast gene mutation assays. In contrast, gene mutation studies in Chinese hamster V79 and CHO cells were positive. Chromosomal aberrations and sister chromatid exchange were also induced by beryllium in cultured human lymphocytes and Syrain hamster embryo cells.

**Bis(2-ethylhexyl)phthalate (BEHP).** BEHP has an estimated oral RfD of 2 x 10<sup>-2</sup> mg/kg/day. This RfD is based on increased liver weight in guinea pigs and rats and includes a total uncertainty factor of 1000. Factors of 10 each were used for interspecies variation and for protection of sensitive human subpopulations. An additional factor of 10 was used since the guinea pig exposure was longer than subchronic but less than lifetime, and because, while the RfD is set on a LOAEL, the effect observed was considered to be minimally adverse. Confidence in this RfD is described as medium.

Male and female guinea pigs consumed feed containing BEHP. No treatment-related effects were observed on mortality, body weight, kidney weight, or gross pathology and histopathology of kidney, liver, lung, spleen, or testes. Statistically significant increases in relative liver weights were observed in treated females. Groups of male and female Sherman rats were fed diets containing BEHP. Mortality in the treated and control groups was high; 46.2 and 42.7%, respectively, survived to 1 year. There was, however, no effect of treatment on either parental or offspring mortality, life expectancy, hematology, or histopathology of organs. Both parental and offspring rats receiving the BEHP diet were retarded in growth and had increased kidney and liver weights.

In additional studies dietary levels of 0, 0.01, 0.1, and 0.3 percent BEHP were administered to male and female CD-1 mice that were examined for adverse fertility and reproductive effects using a continuous breeding protocol. BEHP was a reproductive toxicant in both sexes significantly decreasing fertility and the proportion of pups born alive per litter at the 0.3% level, and inducing damage to the seminiferous tubules. DEHP has been observed to be both fetotoxic and teratogenic.

There is currently no reference air concentrations available for BEHP.

BEHP is classified as a B2 carcinogen with an oral Slope Factor of 1.4 x 10<sup>-2</sup> (mg/kg/day)<sup>-1</sup> (Linearized multistage procedure) based on studies in which orally administered DEHP produced significant dose-related increases in hepatocellular carcinomas and adenomasin male B6C3F mice.

In an NTP study, male and female fisher 344 rats were fed diets containing BEHP. Similarly, groups of male and female B6C3F1 mice were given BEHP in the diet. No clinical signs of toxicity were observed in either rats or mice. A statistically significant increase in the incidence of hepatocellular carcinomas and combined incidence of carcinomas and adenoma were observed in female rats and both sexes of mice. The combined incidence of neoplastic nodules and hepatocellular carcinomas was statistically significantly increased in the high-dose male rats. A positive dose response trend was also noted.

Carpenter did not find a carcinogenic effect in guinea pigs and dogs exposed to BEHP. Both guinea pigs and dogs were terminated after 1 year of exposure. The treatment and survival periods for these animals were considerably below their lifetimes. Human studies are inadequate to show carcinogenicity. Thiess conducted a mortality study of 221 BEHP production workers exposed to unknown concentrations of DEHP for 3 months to 24 years. Workers were followed for a minimum of 5 to 10 years (mean follow-up time was 11.5 years). Eight deaths were reported in the exposed population. Deaths attributable to pancreatic carcinoma (1 case) and uremia (one case in which the workers also had urethral and bladder papillomas) were significantly elevated in workers exposed for >15 years when compared to the corresponding age groups in the general population. The study is limited by a short follow-up period and unquantified worker exposure. Results are considered inadequate for evidence of a causal association.

Studies indicate that DEHP is not a direct acting mutagen in either a forward mutation assay in Salmonella typhimurium or the recassay in Bacillus subtilis. Information is not available on the potential carcinogenicity of BEHP via inhalation.

**Bromodichloromethane.** An RfD of 2 x 10<sup>-2</sup> mg/kg/day and an LOAEL of 17.9 mg/kg/day were reported with an uncertainty factor of 1000. A factor of 100 was employed for extrapolation from animal data and for protection of sensitive human subpopulations. An additional factor of 10 was used because the RfD was based on a LOAEL (although minimally adverse), and to account for data base deficiencies (no reproductive studies).

Bromodichloromethane (BDCM) was administered in corn oil by gavage. Final mean body weights of dosed female mice and high-dose male and female rats were 75 to 91 percent that of vehicle controls.

Compound-related nonneoplastic lesions included cytomegaly and tubular cell hyperplasia of the kidney and fatty metamorphosis of the liver in male rats; eosinophilic cytoplasmic change, clear cell change, focal cellular change, and fatty metamorphosis of the liver and tubular cell hyperplasia of the kidney in female rats; fatty metamorphosis of the liver, renal cytomegaly, and follicular cell hyperplasia of the thyroid gland in male mice; and follicular cell hyperplasia of the thyroid gland in female mice.

In a subchronic bioassay conducted by NTP, male and female rats received doses of Bromodichloromethane. Centrilobular degeneration of the liver and degeneration and necrosis of the kidney were seen in high-dose male rats; liver lesions were observed in high-dose female rats and in female mice at a lower dose, and kidney lesions were seen in male mice at a low dose. These data define a NOAEL of 35.7 mg/kg/day, a dose above which produced kidney lesions and depressed body weight in male mice. Because the chronic study used more animals/dose, was of longer duration, and presented more complete data, more confidence is placed in the chronic LOAEL than in the subchronic NOAEL.

There are no published data on teratogenicity or reproductive effects of trihalomethanes.

Confidence in the study is rated medium because although NTP incorporated both chronic and subchronic exposures in two species using sufficient numbers of animals and measured multiple endpoints, including histopathology of most organ systems, a NOEL was not determined. Although there are some discrepancies in the dose levels producing adverse effects, there are several published subchronic studies of bromodichloromethane permitting confidence in the data base to be rated medium to low. Thus, overall confidence in the RfD is rated medium to low.

Bromodichloromethane is classified as a class B2 probable human carcinogen. This is based on inadequate human data and sufficient evidence of carcinogenicity in two animal species (mice and rats) as shown by increased incidence of kidney tumors and tumors of the large intestine in male and female rats, kidney tumors in male mice, and liver tumors in female mice.

In a 2-year carcinogenicity study, bromodichloromethane was administered in corn oil by gavage. The study using the male rats was restarted at 10.5 months into the original study because a temperature elevation killed 45/50 of the vehicle control male rats. Survival in vehicle control and dosed female mice was reduced after week 84; the mortality was associated with ovarian abscesses.

Bromodichloromethane induced tumors in the large intestine, kidney and/or liver of mice and rats. In male mice, the incidence of tubular cell adenomas and the combined incidence of tubular cell adenomas or adenocarcinomas of the kidneys were statistically significantly increased in the high-dose group.

In female mice, a significant, dose-related increase was observed in the incidence of hepatocellular adenomas or carcinomas (combined); incidences of these tumors were significantly higher than controls for both low-dose and high-dose female mice.

In rats, the incidence of tubular cell adenomas and adenocarcinomas, and the combined incidence of adenomas and adenocarcinomas were statistically significantly increased in male and female rats only in the high-dose group. Tumors of the large intestine, namely adenomatous polyps, adenocarcinomas, and polyps or adenocarcinomas (combined) were significantly increased in males in a dose-dependent manner, whereas these tumors were observed only at the high dose in females. Neoplasms of the large intestine are uncommon in this strain of rats; the historical control incidence of large intestine tumors is less than 0.2% in males and 0 percent in females. Under the conditions of this bioassay, NTP concluded there was clear evidence of carcinogenicity of bromodichloromethane in male and female F344/N rats and B6C3F1 mice.

Theiss tested bromodichloromethane in a short-term lung adenoma test in strain A/St mice. There was no effect of treatment on survival. Twenty-four weeks after the first injection, the mice were sacrificed and the lungs examined for surface adenomas. The number of pulmonary tumors per mouse was elevated in the high-dose animals, although the increase was not statistically significant.

Tumasonis administered 1.2 mL bromodichloromethane per liter of drinking (tap) water to male and female Wistar rats for 72 weeks, after which concentrations were halved for the remainder of the lifetime of the animals (140 to 180 weeks). Controls were untreated. Body weight was decreased in treated animals relative to controls by approximately 35 to 40%.

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Hepatic neoplastic nodules were statistically significantly elevated in female rats when compared with controls. Neoplastic nodules in males and lymphosarcomas and pituitary tumors in both sexes were reported but did not have an increased incidence relative to the controls. Two males and one female were noted to have renal adenoma or adenocarcinoma, while none were reported in the control group.

Voronin examined the carcinogenicity of bromodichloromethane in CBA x C57Bl/6 mice. The authors concluded that under the conditions of this bioassay bromodichloromethane was not carcinogenic.

Bromodichloromethane was mutagenic in Salmonella typhimurium strains TA100 and TA1535 in both the presence and absence of liver homogenate in a vapor phase test performed in a desiccator. When tested in a standard Salmonella/microsomal assay, however, the compound was not mutagenic in both the presence and absence of liver homogenate in strains TA98, TA100, TA1535, TA1537, and TA1538. Mortelmans reported bromodichloromethane to be not mutagenic in S. typhimurium strains TA98, TA100, TA1535, or TA1538 both with and without rat or hamster liver homogenate. Simmon reported a mutagenic effect in Escherichia coli WP2 exposed to bromodichloromethane both with and without liver homogenate in a desiccator. Bromodichloromethane did not induce mitotic recombination in the presence or absence of liver homogenate in studies with Saccharomyces cerevisiae strain D3. However, Nestmann and Lee observed weak effects in S. cerevisiae strains D7 and XV185-14C following exposure to bromodichloromethane in the absence of liver homogenate.

Bromodichloromethane was not mutagenic in the mouse lymphoma L5178/TK+/- assay in the absence of rat liver homogenate but did induce forward mutations in this system in the presence of rat liver homogenate. Morimoto and Koizumi reported that bromodichloromethane produced a significant increase in the frequency of sister chromatid exchanges (SCEs) in both cultured human peripheral blood lymphocytes treated in vitro and mouse bone marrow cells treated in vivo. NTP reported that cytogenetic tests with Chinese hamster ovary cells demonstrated no induction of chromosomal aberrations or SCEs following treatment with bromodichloromethane in either the presence or absence of liver homogenate. Bromodichloromethane is structurally similar to other known animal carcinogens such as dibromochloromethane and chloroform.

**Bromoform.** An oral RfD of 2 x 10<sup>-2</sup> mg/kg/day, an NOAEL of 17.9 mg/kg/day, and an LOAEL of 35.7 mg/kg/day were reported with an uncertainty factor of 1000. Factors of 10

each were employed for use of a subchronic assay, for extrapolation from animal data, and for protection of sensitive human subpopulation.

In a study by NTP (National Toxicology Program) rodents were subjected to various concentrations of bromoform. Liver histology was conducted on all rats and on male mice receiving doses greater than 100 mg/kg. Females of both species did not show any chemically-related effects. A decrease in body weight of both sexes of mice was reported, but was not dose-related. The male mice showed fatty metamorphosis of the liver at doses of 200 and 400 mg/kg. The only effect reported for male rats was a dose-related increase in clear cell foci of the liver. A Fisher Exact Test showed that the incidence of the clear cell foci at doses of 50 mg/kg (the LOAEL) or above was statistically elevated relative to the vehicle control, therefore, 25 mg/kg is the NOEL for F344/N rats.

There are no adequate published data on teratogenicity or reproductive effects of trihalomethanes.

The NTP study utilized both sexes of two species of animals. Both species showed liver lesions, but the study did not investigate clinical chemistries or perform urinalysis; thus, confidence in the study is rated medium. Several studies support the choice of hepatic lesions as the critical effect for the basis of the RfD, but the chosen study is of subchronic duration and reproductive effects have not been monitored; thus, the data base is rated medium to low. Medium to low confidence in the RfD follows.

The 1985 Drinking Water Criteria Document for Trihalomethanes is currently undergoing Agency review.

Bromoform is classified as a B2 probable human carcinogen-based on inadequate human data and sufficient evidence of carcinogenicity in animals, namely an increased incidence of tumors after oral administration of bromoform in rats and intraperitoneal administration in mice.

Bromoform is genotoxic in several assay systems. Also, bromoform is structurally related to other trihalomethanes (e.g., chloroform, bromodichloromethane, dibromochloromethane) which have been verified as either probable or possible carcinogens.

Human carcinogenicity data is inadequate. Cantor suggests a positive correlation between levels of trihalomethane in drinking water and the incidence of several human cancers. Additional geographic studies of bromoform indicate that there may be an association between the levels of trihalomethanes in drinking water and the incidence of bladder, colon, rectal, or pancreatic cancer in humans. However, the information from these studies is considered incomplete and preliminary because their designs do not permit consideration of several possible variables which may be involved (e.g., personal habits, information on residential histories, and past exposures).

Bromoform has been tested for animal carcinogenicity in two species, rat and mouse, by oral or intraperitoneal administration.

Neoplastic lesions (adenomatous polyps or adenocarcinomas) were observed in the large intestine (colon or rectum) of male rats and female rats. Adenocarcinomas alone were not significantly increased compared with controls. The reduced survival of male rats in the high-dose group may account for the lower incidence of lesions in this group. No treatment-related tumors were observed in mice at either dose level. Under the conditions of this study, the NTP judged there was clear evidence of carcinogenicity for female rats, some evidence of carcinogenicity for male rats, and no evidence of carcinogenicity for male and female mice.

In a feeding study with microencapsulated bromoform, Kurokawa observed no evidence of carcinogenicity in male or female Wistar rats exposed for 24 months at various concentrations.

Pereira determined that bromoform did not induce GGTase-positive foci in the rat liver at 1 mM (253 mg/kg) or 0.8 mM (202 mg/kg) following a 2/3 hepatectomy and promotion with phenobarbital. However, Pereira found that bromoform is a potent inducer of ornithine decarboxylase, which is an indication of tumor promotion activity in the skin and liver.

Bromoform has been shown to produce mutations in Salmonella typhimurium strains TA97, TA98, TA100, and TA1535 with and without rat hepatic homogenates. Bromoform also produces mutations at the TK locus in mouse cells; SCE induction in Chinese hamster ovary cells, human lymphocytes (in vitro) and mouse bone marrow cells (in vivo); chromosomal aberrations in Chinese hamster ovary cells; cell cycle delay in human lympho-

cytes; and an increased incidence of micronuclei in bone marrow erythrocytes from mice given bromoform i.p.

**Cadmium.** An oral RfD of 5 x  $10^{-4}$  mg/kg/day, a NOAEL of 0.005 mg/kg/day and 0.01 mg/kg/day for water and food respectively, were reported with an uncertainty factor of 10. This uncertainty factor is used to account for intrahuman variability to the toxicity of this chemical in the absence of specific data on sensitive individuals.

A concentration of 200 ug cadmium (Cd)/gm wet human renal cortex is the highest renal level not associated with significant proteinuria. A toxicokinetic model is available to determine the level of chronic human oral exposure (NOAEL) which results in 200 ug Cd/gm wet human renal cortex; the model assumes that 0.01 percent day of the Cd body burden is eliminated per day. Assuming 2.5 percent absorption of Cd from food or 5 percent from water, the toxicokinetic model predicts that the NOAEL for chronic Cd exposure is 0.005 and 0.01 mg Cd/kg/day from water and food, respectively (i.e., levels which would result in 200 ug Cd/gm wet weight human renal cortex). Thus, based on an estimated NOAEL of 0.005 mg Cd/kg/day for Cd in drinking water and an UF of 10, an RfD of 0.0005 mg Cd/kg/day (water) was calculated; an equivalent RfD for Cd in food is 0.001 mg Cd/kg/day.

Cd is unusual in relation to most, if not all, of the substances for which an oral RfD has been determined in that a vast quantity of both human and animal toxicity data are available. The RfD is based on the highest level of Cd in the human renal cortex (i.e., the critical level) not associated with significant proteinuria (i.e., the critical effect). A toxicokinetic model has been used to determine the highest level of exposure associated with the lack of a critical effect. Since the fraction of ingested Cd that is absorbed appears to vary with the source (e.g., food vs. drinking water), it is necessary to allow for this difference in absorption when using the toxicokinetic model to determine an RfD.

The choice of NOAEL does not reflect the information from any single study. Rather, it reflects the data obtained from many studies on the toxicity of cadmium in both humans and animals. These data also permit calculation of pharmacokinetic parameters of cadmium absorption, distribution, metabolism and elimination. All of this information considered together gives high confidence in the data base. High confidence in either RfD follows as well.

A risk assessment for this substance/agent is under review by an EPA work group.

Cadmium is a class B1 probable human carcinogen based on a limited evidence from occupational epidemiologic studies of cadmium is consistent across investigators and study populations. There is sufficient evidence of carcinogenicity in rats and mice by inhalation and intramuscular and subcutaneous injection. Seven studies in rats and mice wherein cadmium salts (acetate, sulfate, chloride) were administered orally have shown no evidence of carcinogenic response.

A 2-fold excess risk of lung cancer was observed in cadmium smelter workers. The cohort consisted of 602 white males who had been employed in production work a minimum of 6 months during the years 1940-1969. The population was followed to the end of 1978. Urine cadmium data available for 261 workers employed after 1960 suggested a highly exposed population. The authors were able to ascertain that the increased lung cancer risk was probably not due to the presence of arsenic or to smoking. An evaluation by the Carcinogen Assessment Group of these possible confounding factors has indicated that the assumptions and methods used in accounting for them appear to be valid. As the SMRs observed were low and there is a lack of clear cut evidence of a causal relationship of the cadmium exposure only, this study is considered to supply limited evidence of human carcinogenicity.

An excess lung cancer risk was also observed in three other studies which were, however, compromised by the presence of other carcinogens (arsenic, smoking) in the exposure or by a small population.

Four studies of workers exposed to cadmium dust or fumes provided evidence of a statistically significant positive association with prostate cancer, but the total number of cases was small in each study. The Thun study is an update of an earlier study and does not show excess prostate cancer risk in these workers. Studies of human ingestion of cadmium are inadequate to assess carcinogenicity.

Exposure of Wistar rats by inhalation to cadmium as cadmium chloride at concentrations of 12.5, 25 and 50 ug/cu.m for 18 months, with an additional 13-month observation period, resulted in significant increases in lung tumors. Intratracheal instillation of cadmium oxide did not produce lung tumors in Fischer 344 rats but rather mammary tumors in males and tumors at multiple sites in males. Injection site tumors and distant site tumors (for example, testicular) have been reported by a number of authors as a consequence of intramuscular or subcutaneous administration of cadmium metal and chloride, sulfate, oxide and sulfide compounds of cadmium to rats and mice. Seven studies in rats and mice where cadmium

salts (acetate, sulfate, chloride) were administered orally have shown no evidence of a carcinogenic response.

Results of mutagenicity tests in bacteria and yeast have been inconclusive. Positive responses have been obtained in mutation assays in Chinese hamster cells (Dom and V79 lines) and in mouse lymphoma cells.

Conflicting results have been obtained in assays of chromosomal aberrations in human lymphocytes treated in vitro or obtained from exposed workers. Cadmium treatment in vivo or in vitro appears to interfere with spindle formation and to result in aneuploidy in germ cells of mice and hamsters.

**Chloroform.** An oral RfD of 1x10 mg/kg/day and an LOAEL of 12.9 mg/kg/day with an uncertainty factor of 1000. Uncertainty factors of 10 each were applied to the LOAEL of 12.9 mg/kg/day to account for the interspecies conversion, protection of sensitive human subpopulations, and concern that the effect seen was a LOAEL and not a NOEL.

Beagle dogs were administered chloroform in a toothpaste base in gelatin capsules. Fatty cysts, considered to be treatment-related, were observed in livers of some dogs in both treatment groups. Nodules of altered hepatocytes were considered treatment-related but not dose-dependent. A dose-related increase in SGPT levels was noted and a less marked increase in SGOT was noted in the high-dose animals. The LOAEL was determined to be 12.9 mg/kg/day, and an RfD was set at 0.01 mg/kg/day.

Chloroform is considered to be highly fetotoxic, but not teratogenic.

A study in rats, using only one treatment dose, identified 60 mg/kg/day by gavage as a LOAEL for decreased weight gain, plasma cholinesterase and relative liver weight. Other data in the literature also indicate changes in liver fat to be treatment-related.

The critical study was of chronic duration, used a fairly large number of dogs, and measured multiple endpoints; however, only two treatment doses were used and no NOEL was determined. Therefore, confidence in the study is rated medium. Confidence in the data base is considered medium to low; several studies support the choice of a LOAEL, but a NOEL was not found. Confidence in the RfD is also considered medium to low.

The 1985 Drinking Water Criteria Document for Trihalomethanes is currently undergoing Agency review.

A risk assessment for this substance/agent is under review by an EPA work group.

Chloroform is considered a class B2 probable human carcinogen based on increased incidence of several tumor types in rats and three strains of mice.

Human carcinogenicity information is inadequate. There are no epidemiologic studies of chloroform itself. Chloroform and other trihalomethanes are formed from the interaction of chlorine with organic material found in water. Several ecological and case-control studies of populations consuming chlorinated drinking water in which chloroform was the major chlorinated organic show small significant increases in the risk of rectal bladder or colon cancer on an intermittent basis. Many other suspected carcinogens were also present in these water supplies.

Chloroform has been tested for carcinogenicity in eight strains of mice, two strains of rats and in beagle dogs. In a gavage bioassay, Osborne-Mendel rats and B6C3F1 mice were treated with chloroform in corn oil. A significant increase in kidney epithelial tumors was observed in male rats and highly significant increases in hepatocellular carcinomas in mice of both sexes. Liver nodular hyperplasia was observed in low-dose male mice not developing hepatocellular carcinoma. Hepatomas have also developed in female strain A mice and NLC mice gavaged with chloroform.

Jorgenson administered chloroform (pesticide quality and distilled) in drinking water to male Osborne-Mendel rats and female B6C3F1 mice. A significant increase in renal tumors in rats was observed in the highest dose group. The increase was dose related. The liver tumor incidence in female mice was not significantly increased. This study was specifically designed to measure the effects of low doses of chloroform.

Chloroform administered in toothpaste was not carcinogenic to male C57B1, CBA, CF-1 or female ICI mice or to beagle dogs. Male ICI mice administered were found to have an increased incidence of kidney epithelial tumors. A pulmonary tumor bioassay in strain A/St mice was negative as was one in which newborn C57X DBA2/F1 mice were treated s.c. on days 1 to 8 of life.

The majority of tests for genotoxicity of chloroform have been negative. These negative findings include covalent binding to DNA, mutation in Salmonella, a Drosophila sex-linked recessive, tests for DNA damage a micro-nucleus test, and transformation of BHK cells. By contrast one study demonstrated binding of radiolabeled chloroform to calf thymus DNA following metabolism by rat liver microsomes. Chloroform caused mitotic recombination in Saccharomyces and sister chromatid exchange in cultured human lymphocytes and in mouse bone marrow cells exposed in vivo.

The carcinogenicity of chloroform may be a function of its metabolism to phosgene, which is known to cross-link DNA. A host-mediated assay using mice indicated that chloroform was metabolized in vivo to a form mutagenic to Salmonella strain TA1537. Likewise urine extracts from chloroform-treated mice were mutagenic.

Chloroform administered to mice in drinking water promoted growth and metastasis of Ehrlich ascites cells injected i.p.

**Chromium** (III). An oral RfD of 1.0 mg/kg/day and an NOEL of 1800 g/kg with an uncertainty factor of 100. The factor of 100 represents two 10-fold decreases in mg/kg bw/day dose that account for both the expected interhuman and interspecies variability to the toxicity of the chemical in lieu of specific data.

Ivankovic, S. and R. Preussmann subjected rats to food contaminated with chromic oxide (Cr2O3) baked in bread. No effects due to Cr2O3 treatment were observed at any dose level.

Ivankovic and Preussmann also treated rats at higher dietary levels of Cr2O3. The only effects observed were reductions in the absolute weights of the livers and spleens of animals in the high-dose group. Organ weights relative to body weight were not reported.

Other subchronic oral studies show no indication of adverse effects attributable to trivalent chromium compounds, but dose levels were considerably lower.

This RfD is limited to metallic chromium (III) of insoluble salts. Examples of insoluble salts include chromic III oxide (Cr203) and chromium III sulfate [Cr2(SO4)3].

Very limited data suggest that Cr III may have respiratory effects on humans. No data on chronic or subchronic effects of inhaled Cr III in animals can be found. Adequate teratology data do not exist, but reproductive effects are not seen at dietary levels of 5 percent Cr2O3.

The principal study is rated low because of the lack of explicit detail on study protocol and results. Low confidence in the data base reflects the lack of high-dose supporting data. The low confidence in the RfD reflects the foregoing, but also reflects the lack of an observed effect level. Thus, the RfD, as given, should be considered conservative, since the MF addresses only those factors which might lower the RfD.

A risk assessment for this substance/agent is under review by an EPA work group.

Rats were exposed to drinking water containing Cr(VI) (K2CrO4) at levels of 80 or 134 mg Cr(VI)/L for 60 days (8.3 or 14.4 mg Cr(VI)/kg/day, respectively) without adverse effects. Therefore, a NOAEL of 14.4 mg/kg/day is identified.

In a 1-year drinking water study, consumption of water containing either Cr(III) (CrCl3) or Cr(VI) (K2CrO4) (0 to 1.87 mg/kg/day for male rats and 0 to 2.41 mg/kg/day for female rats) produced no significant differences in weight gain, appearance, or pathological changes in the blood or other tissue. Therefore, a NOAEL of 2.41 mg/kg/day is identified.

**Chromium (VI).** An oral RfD of 5 x 10<sup>-3</sup> mg/kg/day and an NOAEL of 2.4 mg/kg/day with an uncertainty factor of 500. The uncertainty factor of 500 represents two 10-fold decreases in dose to account for both the expected interhuman and interspecies variability in the toxicity of the chemical in lieu of specific data, and an additional factor of 5 to compensate for the less-than-lifetime exposure duration of the principal study.

MacKenzie, R.D., R.U. Byerrum, C.F. Decker, C.A. Hoppert and R.F. Langham treated groups of Sprague-Dawley rats with drinking water containing hexavalent chromium (as K2CrO4) for 1 year. No significant adverse effects were seen on appearance, weight gain, or food consumption, and there were no pathologic changes in the blood or other tissues in any treatment group. An abrupt rise in tissue chromium concentrations was noted in rats treated with greater than 5 ppm. The authors stated that "apparently, tissues can accumulate considerable quantities of chromium before pathological changes result." In the 25 ppm treatment groups, tissue concentrations of chromium were approximately 9 times higher for those treated with hexavalent chromium than for the trivalent group.

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Similar no-effect levels have been observed in dogs and humans. Anwar observed no significant effects in female dogs (2/dose group) given chromium(VI) (as K2CrO4) in drinking water for 4 years.

This RfD is limited to metallic chromium(VI) of soluble salts. Examples of soluble salts include potassium dichromate (K2CR2O7), sodium dichromate (Na2Cr2O7), potassium chromate (K2CrO4) and sodium chromate (Na2CrO4).

Trivalent chromium is an essential nutrient. There is some evidence to indicate that hexavalent chromium is reduced in part to trivalent chromium in vivo.

The literature available on possible fetal damage caused by chromium compounds is limited. No studies were located on teratogenic effects resulting from ingestion of chromium.

Confidence in the chosen study is low because of the small number of animals tested, the small number of parameters measured and the lack of toxic effect at the highest dose tested. Confidence in the data base is low because the supporting studies are of equally low quality, and teratogenic and reproductive endpoints are not well studied. Low confidence in the RfD follows.

A risk assessment for this substance/agent is under review by an EPA work group. Chromium VI is classified as a class A human carcinogen due to the results of occupational epidemiologic studies of chromium-exposed workers are consistent across investigators and study populations.

Epidemiologic studies of chromate production facilities in the United States, Great Britain, Japan, and West Germany have established an association between chromium (Cr) exposure and lung cancer. Most of these studies did not attempt to determine whether Cr III or Cr VI compounds were the etiologic agents.

Three studies of the chrome pigment industry, one in Norway, one in England, and the third in the Netherlands and Germany also found an association between occupational chromium exposure (predominantly to Cr VI) and lung cancer.

Results of two studies of the chromium plating industry were inconclusive, while the findings of a Japanese study of chrome platers were negative. The results of studies of ferrochromium workers were inconclusive as to lung cancer risk.

Hexavalent chromium compounds were carcinogenic in animal assays producing the following tumor types: intramuscular injection site tumors in Fischer 344 and Bethesda Black rats and in C57BL mice; intra-plural implant site tumors for various chromium VI compounds in Sprague-Dawley and Bethesda Black rats; intrabronchial implantation site tumors for various Cr VI compounds in Wistar rats; Levy as quoted in NIOSH); and subcutaneous injection site sarcomas in Sprague-Dawley rats.

A large number of chromium compounds have been assayed in in vitro genetic toxicology assays. In general, hexavalent chromium is mutagenic in bacterial assays whereas trivalent chromium is not. Likewise Cr VI but not Cr III was mutagenic in yeasts and in V79 cells. Chromium III and VI compounds decrease the fidelity of DNA synthesis in vitro, while Cr VI compounds inhibit replicative DNA synthesis in mammalian cells and produce unscheduled DNA synthesis, presumably repair synthesis, as a consequence of DNA damage. Chromate has been shown to transform both primary cells and cell lines. Chromosomal effects produced by treatment with chromium compounds have been reported by a number of authors; for example, both Cr VI and Cr III salts were clastogenic for cultured human leukocytes.

There are no long-term studies of ingested Cr VI. There appears to be significant in vivo conversion of Cr VI to Cr III and III to VI; Cr III is an essential trace element.

1,2-Dichlorobenzene. An oral Rfd of 9 x 10<sup>-2</sup> and an NOAEL of 120 mg/kg/day with an uncertainty factor of 1000 was reported that allows for uncertainty in the extrapolation of dose levels from laboratory animals to humans (10A), uncertainty in the threshold for sensitive humans (10H), and uncertainty because of the lack of studies assessing reproductive effects and adequate chronic toxicity in a second species (10D). The chronic study, coupled with results of subchronic studies provides a NOAEL and LOAEL for several toxicologic endpoints, but the chronic study did not assess biochemical and clinical endpoints. Therefore, a medium level of confidence is assigned. Lack of reproductive and adequate additional supporting toxicity studies in nonrodent species lead to low confidence in the data base.

1,2-Dichlorobenzene in corn oil was given by gavage to F344/N rats and B6C3F1 mice. An increase in renal tubular regeneration in high-dose male mice was observed there was no other evidence of treatment-related renal lesions in either species. Further, the incidence of this lesion in male control mice was below those of three similar control groups that were studied during approximately the same period at the testing facility. There was no other evidence of treatment-related effects in this study. Because the decrease in survival and the increase in renal tubular regeneration in the high-dose animals were of questionable significance, a NOAEL of 120 mg/kg/day is established.

1,2-Dichlorobenzene in corn oil was given orally by gavage to F344/N rats and B6C3F1 mice. Liver necrosis was found in mice and rats given 250 mg/kg/day. Deaths, degeneration and necrosis in the liver, lymphocyte depletion in the spleen and thymus, renal tubular degeneration (male rats only), and slight decreases in hemoglobin, hematocrit and red blood cell counts (rats only) were induced at 500 mg/kg/day.

Hepatocellular necrosis (focal or individual hepatocyte) was observed in 1 male and 3 female rats given 125 mg/kg/day. Increases in serum cholesterol at all doses except 60 mg/kg/day in male rats and at doses of 125 to 500 mg/kg/day for female rats; liver weight/body weight ratios in male and female rats at 125 to 500 mg/kg/day; and serum protein at all doses in female rats and at 250 to 500 mg/kg/day in male rats indicate treatment-related liver effects at doses >125 mg/kg/day. However, no evidence of treatment-related liver pathology in rats and mice given 60 or 120 mg/kg/day, 5 days/week in the 2-year NTP carcinogenicity bioassay and no increase (P=>0.05) in serum enzymes (SGPT, GGPT, alkaline phosphatase) for either rats or mice in the 13-week study are grounds for considering 125 mg/kg/day as a NOAEL in the 13-week study.

In rats dosed by gavage with 1,2-dichlorobenzene at 18.8, 188, or 376 mg/kg/day, 5 days/week for 192 days, liver and kidney weights were increased at 188 mg/kg/day, and liver pathology and increased spleen weight were observed at 376 mg/kg/day. No effects were observed at 18.8 mg/kg/day. Thus, the NOAEL was 18.8 mg/kg/day.

Rats, guinea pigs, mice, rats, and monkeys were exposed by inhalation to 1,2-dichlorobenzene at levels of 49 or 93 ppm, 7 hours/day, 5 days/week for 6 to 7 months. At 93 ppm, body weight gain in rats and spleen weight in guinea pigs were reduced (P=>0.05). Estimated daily doses with 49 ppm exposure are 387 mg/kg (mouse), 19.3 mg/kg (rat), 14.4 mg/kg (guinea pig), 15.9 mg/kg (rabbit), and 20.3 mg/kg (monkey).

Pregnant F344/N rats and New Zealand rabbits were exposed by inhalation to 0, 100, 200, or 400 ppm 1,2-dichlorobenzene 6 hours daily on days 6 through 15 (rats) or 6 through 18 (rabbits) of gestation. Body weight gain was lower (P=>0.05) in rats at all doses and in rabbits at 400 ppm, during the first 3 days of exposure. Liver weights (absolute and relative to body weight) were increased in rats at 400 ppm. No developmental toxicity was evident at any dose. Estimated daily doses at 100 ppm exposure are 40 mg/kg (rat) and 32 mg/kg (rabbit).

1,2-Dichlorobenzene is classified as a class D carcinogen due to a lack of human data and both negative and positive trends for carcinogenic responses in rats and mice. It is also classified as a class "C" pesticide.

Two carcinogenicity studies were conducted by the National Toxicology Program using 1,2-dichlorobenzene. Survival was statistically significantly reduced in the high-dose males due to causes incidental to treatment. An increased incidence of pheochromocytomas of the adrenal gland was found in low-dose males but not the high-dose males. The increased incidence of pheochromocytomas in low-dose males was discounted because there was no dose-response trend or high-dose effect, no malignant pheochromocytomas had been observed, and no incidence increase was seen in females; additionally, the biological consequence of this endpoint is often questioned because pheochromocytomas are not considered to be a life-threatening condition. There was a decrease in the incidence of testicular interstitial cell tumors.

In the mouse study, no significant differences in survival were noted in the treatment groups when compared with controls. A dose-related increase was seen in malignant histiocytic lymphoma in male mice and female mice. An increased incidence of alveolar and bronchiolar carcinomas (combined) in male mice was significant by a trend test; the combined incidence of alveolar and bronchiolar adenomas and carcinomas did not show a significant elevation. One high-dose male had a testicular interstitial cell tumor. In males there was a decrease in the incidence of hepatocellular adenomas and carcinomas.

Chromosome studies, in workers occupationally exposed for 4 days (8 hours/day) to 1,2-dichlorobenzene vapors showed a statistically significant increase in the incidence of chromosomal alterations in chromosomes isolated from peripheral blood cells. The number of

single and double chromosome breaks was also increased. A followup study 6 months after the initial exposure indicated a significant increase in only double chromosome breaks.

1,1-Dichloroethane. A risk assessment for inhalation RfD this substance/agent is under review by an EPA work group.

1,1-Dichloroethane is classified as a C possible human carcinogen based on no human data and limited evidence of carcinogenicity in two animal species (rats and mice) as shown by an increased incidence of mammary gland adenocarcinomas and hemangiosarcomas in female rats and an increased incidence of hepatocellular carcinomas and benign uterine polyps in mice.

An NCI bioassay provides limited evidence of the carcinogenicity of 1,1-dichloroethane in Osborne-Mendel rats and B6C3F1 mice. This is based on significant dose-related increases in the incidence of hemangiosarcomas at various sites and mammary carcinomas in female rats and statistically significant increases in the incidence of liver carcinoma in male mice and benign uterine polyps in female mice. The study is limited by high mortality in many groups; the low survival rates precluded the appearance of possible late-developing tumors and decreased the statistical power of this bioassay.

Technical grade 1,1-dichloroethane in corn oil was administered by gavage. In female rats there was a statistically significant positive dose-related trend in incidence of hemangiosarcomas. The incidence of mammary gland adenocarcinomas showed a statistically significant dose-related positive trend in those female rats surviving at least 52 weeks; tumor incidence increased respectively with the dosage. No mammary gland adenomas or hemangiosarcomas were observed in the dosed-male rats.

In the same NCI study, groups of 50 B6C3F1 mice/sex/group were administered technical grade 1,1-dichloroethane in corn oil by gavage. An increased incidence of hepatocellular carcinoma in male mice was not statistically significant by either pair-wise or trend test. The incidence of hepatocellular carcinoma in male mice was displayed a positive trend that was statistically significant. In female mice, liver carcinomas were reported in only the vehicle control and the low-dose groups: no liver tumors were seen in the untreated controls or in the high-dose group. A statistically significant increase in benign uterine endometrial stromal polyps was observed in high-dose females; these were not observed in any other group. A preliminary report of the NCI study was published by Weisburger.

To determine if 1,1-dichloroethane in drinking water could act as a tumor promoter or a complete carcinogen, Klaunig exposed groups of 35 male B6C3F1 mice to 1,1-dichloroethane in drinking water. Neither the initiated nor the noninitiated 1,1-dichloroethane-treated groups showed a significant increase in the incidence of liver or lung tumors compared with initiated or noninitiated controls, respectively. The authors concluded that 1,1-dichloroethane was not carcinogenic to mice and did not act as a tumor promotor following initiation with DENA. These conclusions may not be entirely justified, since the duration of the study may have been inadequate for the development of tumors in noninitiated 1,1-dichloroethane-treated animals. In addition, the incidence of liver tumors in DENA-initiated controls was 70% at 24 weeks and 100 percent at 52 weeks, and the number of tumors/mouse in DENA-initiated controls at these times was 3.00 and 29.30, respectively. Hence, an increase in tumors or decrease in latency in 1,1-dichloroethane-treated DENA-initiated animals would have to be marked in order to be detectable.

Milman and Story investigated the chlorinated ethanes and ethylenes to detect their potential tumor initiating or promoting effects in a liver foci assay in Osborne-Mendel rats. In this assay, 1,1-dichloroethane did not give positive results for initiation (with phenobarbital as promotor), or as a complete carcinogen when administered in the absence of initiation or promotion. Positive results for were seen for promotion with DENA as initiator. The assumption that the liver foci seen in this type of assay are precancerous has not been validated.

Lattanzi determined that 1,1-dichloroethane, like 1,2-dichloroethane, binds covalently to DNA, forming DNA adducts. The Covalent Binding Index (CBI) of both 1,1-dichloroethane and 1,2-dichloroethane classifies them as weak initiators.

Chronic bioassays performed by NCI on the isomer 1,2-dichloroethane resulted in many of the same tumor types as seen in the bioassays of 1,1-dichloroethane. Significant increases in the incidences of forestomach squamous cell carcinomas and hemangiosarcomas were observed in male rats and an increased incidence of mammary adenocarcinomas was observed in both female rats and mice. In addition, alveolar and bronchiolar adenomas were reported in male and female mice; endometrial stromal polyps and sarcomas in female mice; and hepatocellular carcinomas in male mice.

Based on these findings, as well as the appearance of lung papillomas in mice after topical treatment, 1,2-dichloroethane was classified as a group B2 chemical, a probable human

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carcinogen. Because of similarities in structure and target organs, the carcinogenic evidence for 1,2-dichloroethane is considered to be supportive of the classification of 1,1-dichloroethane in group C, a possible human carcinogen.

**1,2-Dichloroethane.** 1,2-Dichloroethane is classified as a B2 carcinogen, based on the induction of several tumor types in rats and mice treated by gavage and lung papillomas in mice after topical application.

1,2-Dichloroethane in corn oil was administered by gavage to groups of 50 each male and female Osborne-Mendel rats and B6C3F1 mice. All high-dose male rats died after 23 weeks of observation; the last high-dose female died after 15 weeks. Male rats had significantly increased incidence of forestomach squamous-cell carcinomas and circulatory system hemangiosarcomas. Female rats and mice were observed to have significant increases in mammary adenocarcinoma incidence. Mice of both sexes developed alveolar/bronchiolar adenomas, females developed endometrial stromal polyps and sarcomas, and males developed hepatocellular carcinomas.

Inhalation exposure of Wistar, Sprague-Dawley rats and Swiss mice did not result in increased tumor incidence. An elevation that was not statistically significant in lung adenomas was seen in A/st mice treated i.p. with 1,2-dichloroethane in tricaprylin. ICR/Ha Swiss mice treated topically had a significant increase in benign lung papillomas, but not skin carcinomas.

1,2-Dichloroethane was mutagenic for Salmonella in assays wherein excessive evaporation was prevented; exogenous metabolism by mammalian systems enhanced the response. Both somatic cell mutations and sex-linked recessives were induced in Drosphila. Metabolites of 1,2-dichloroethane have been shown to form adducts with DNA after in vitro or in vivo exposures.

An oral slope factor of 9.1 x 10<sup>-2</sup> per (mg/kg)/day was reported for one experiment.

Adequate numbers of animals were treated and observed for the majority of their expected lifespan. The incidence of hemangiosarcoma was significantly elevated in the treated animals and was dose-related. A slope factor of 6.2 x  $10^{-2}$  (mg/kg)/day, calculated from data on hepatocellular carcinomas in male mice, is supportive of the risk estimate.

Reitz found the major urinary metabolites in rats of ingested and inhaled 1,2-dichloroethane to be identical and generated in the same relative amounts.

Appropriate data for calculating a One-day HA are not available. It is recommended that the Longer-term HA for the 10-kg child of 0.74 mg/L be used as the One-day HA. A NOAEL of 7.4 mg/kg/day with an uncertainty factor of 100 that allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study.

A combination of three inhalation studies in which various animal species were exposed to 1,2-dichloroethane for up to 8 months was considered appropriate to use in calculating Longer-term HAs. In these studies, exposures of rats and guinea pigs to air containing 100 ppm 1,2-dichloroethane for 6 to 7 hours/day, 5 days/week resulted in no mortality and no adverse effects as determined by general appearance, behavior, growth, organ function, or blood chemistry. However, similar exposures of rats, guinea pigs, rabbits, and monkeys to air containing 400 or 500 ppm 1,2-dichloroethane resulted in high mortality and varying pathologic findings including pulmonary congestion; diffused myocarditis; slight to moderate fatty degeneration of the liver, kidney, adrenal gland, and heart; and increased plasma prothrombin time. The NOAEL is identified as 100 ppm. Based on the dosing regimen and an assumed absorption factor of 30 percent, this dose is equivalent to 7.4 mg/kg/day.

1,2-Dichloroethane is a Group B2 probable human carcinogen and is structurally similar to ethylene dibromide, a potent carcinogen. Therefore, neither a DWEL nor a Lifetime HA have been calculated for 1,2-dichloroethane.

Treatment technologies which will remove 1,2-dichloroethane from water include granular activated carbon adsorption, air stripping, and boiling.

cis-1,2-Dichloroethylene. A risk assessment for this substance/agent is under review by an EPA work group.

Cis-1,2-Dichloroethylene is classified as a D carcinogen based on no data in humans or animals and generally nonpositive results in mutagenicity assays.

Cis-1,2-Dichloroethylene did not yield positive results for a Salmonella typhimurium spot test assay in the absence of mammalian liver homogenates; however, this compound did cause a

dose-dependent increase in mutations in a host-mediated assay. Cis-1,2-Dichloroethylene at a medium concentration of 2.9 mM produced no positive results in a mutagenicity assay for Escherichia coli K12. Galli reported no positive results for cis-1,2-dichloroethylene in point mutation, mitotic gene conversion and mitotic recombination assays (all for Saccharomyces cerevisiae). In addition, it did not yield positive results in an in vivo (intravenous) host-mediated mutagenicity assay. Cis-1,2-Dichloroethylene did not induce chromosomal aberrations in mouse bone marrow in vivo.

**2,4-Dimethylphenol.** An oral RfD of 2 x 10<sup>-2</sup>, a LOAEL of 250 mg/kg/day, and an NOAEL of 50 mg/kg/day with an uncertainty factor of 300. In another study, an uncertainty factor of 3000 was established: 10 each for inter- and intraspecies variability and 30 for lack of chronic toxicity data, data in a second species and reproductive/developmental studies.

2,4-Dimethylphenol was administered daily to male and female albino mice by gavage. No significant differences were found between treated and vehicle control groups in mean body weight, body weight gains, food consumption, or eye examinations at any dosage. Toxicologically relevant clinical signs were observed only after week 6 in the high-dose groups of both genders included: squinting, lethargy, prostration, and ataxia, with onset shortly after dosing. Statistically significant hematological changes (p < 0.05) included lower mean corpuscular volume and mean corpuscular hemoglobin concentration in females at terminal, but not interim, sacrifice.

At interim sacrifice in female mid- and high-dose groups, blood urea nitrogen (BUN) levels were significantly below vehicle controls; whereas at final sacrifice in the female mid-dose group, BUN levels were significantly higher than vehicle controls. Low-dose males at interim sacrifice had significantly higher cholesterol levels. Significant differences were not found in gross necropsy or histopathological evaluations, or in organ weights, except for an increase in adrenal weights of low-dose females. The LOAEL and NOAEL for this study were 250 and 50 mg/kg/day, respectively.

No other long-term toxicity, reproductive, or developmental studies of 2,4-dimethylphenol were found in the data bases searched. Literature concerning 2,6-dimethylphenol was identified, but an SAR-based RfD is considered inappropriate when a valid long-term toxicity study for 2,4-dimethylphenol is available.

Confidence in the study is medium, since it examined appropriate endpoints and identified both a LOAEL and a NOAEL. The results of this study are consistent with those of a 14-day gavage study. The data base provides no information on chronic and reproductive studies. Low confidence in both the data base and oral RfD follows.

Ethyl Benzene. Ethyl benzene has an estimated RfD of 1 x 10<sup>-1</sup> mg/kg/day. This RfD is based on the toxic effects on growth, mortality, appearance and behavior, hematologic findings, terminal concentration of urea nitrogen in the blood, average organ and body weights, histopathologic findings, and bone marrow counts. An uncertainty factor of 1000 reflects 10 for both intraspecies and interspecies variability to the toxicity of this chemical in lieu of specific data, and 10 for extrapolation of a subchronic effect level to its chronic equivalent. A LOAEL of 408 mg/kg/day is associated with histopathologic changes in liver and kidney. The confidence in this RfD and study is described as low because rats of only one sex were tested and the experiment was not of chronic duration. Confidence in the supporting data base is low because other oral toxicity data were not found. Low confidence in the RfD follows.

An RfC for ethyl benzene is reported as  $1 \times 10^0$  with an uncertainty factor of 300. A NOAEL of 4340 mg/cu.m is reported for inhalation and 434 mg/cu.m for developmental criteria. Inhalation experiments were conducted with Wistar rats and New Zealand white rabbits. In a separate group of rats maternal organs (liver, lungs, kidney, heart, spleen, adrenals, ovaries, and brain) were examined histopathologically one day prior to the end of gestation. Uteri were examined and fetuses were weighed, sexed, and measured for crown-to-rump length, and examined for external, internal and skeletal abnormalities. For statistical analyses, the litter was chosen as the experimental unit.

Ethyl benzene did not elicit embryotoxicity, fetotoxicity, or teratogenicity in rabbits in either experiment. There were no significant incidences of major malformations, minor anomalies, or common variants in fetal rabbits from exposed groups. Maternal toxicity in the rabbits was not evident. There was no evidence of histologic damage in any of the dams' organs. There was a significantly lower number of live kits per liver in the two groups when evaluated by ANOVA and Duncan's Multiple Range Test.

In rats exposed only during gestation, there were no histopathological effects in any of the maternal organs examined. There was no effect on fertility or on any of the other measures of reproductive status. The principal observation in fetuses was an increased incidence

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(p<0.05) of supernumerary and rudimentary ribs in the high exposure group and an elevated incidence of extra ribs in both the high and 100 ppm groups. Both absolute and relative liver, kidney, and spleen weights were significantly increased in pregnant rats from the 1000 ppm group.

No fetal toxicity was noted at either exposure level. Body weights, placental weights, and sex ratios were within normal limits. Absolute and relative liver and spleen weights were significantly increased in pregnant rats from the 1000 ppm group; only relative kidney weight was increased significantly. There were no histopathological effects in any of the organs examined.

The uncertainty factor of 300 reflects a factor of 10 to protect unusually sensitive individuals, 3 to adjust for interspecies conversion and 10 to adjust for the absence of multigenerational reproductive and chronic studies.

New Zealand rabbits were exposed for to various concentrations of ethyl benzene from gestational days 7 to 20. Maternal weight gain was reported to have decreased and exhibited mild maternal toxicity manifested by reduced weight gain.

Postimplantation loss (percent dead or resorbed fetuses), and exposure-related skeletal retardation were significantly elevated (p<0.05) in rats at all exposure levels with one exception that did show an increased incidence of dead/resorbed fetuses, lower weight of fetuses, and skeletal retarded fetuses. There was a significant (p<0.05) increase in skeletal retardation and fetal resorption in all continuous exposure groups although the concentration-response was shallow.

In rats, hematology parameters were unaffected. Of the liver enzymes evaluated, only serum alkaline phosphatase (SAP) activity was significantly reduced in a concentration-related manner (at 500 ppm and above) for both sexes with a greater sensitivity in females. The significance of this decrease is not clear since in liver damage, SAP levels usually increase. The investigators suggested the decrease may be due to reduced water and food intake. No liver histopathology was noted for any exposure group. Regeneration of renal tubules in the kidneys of male rats only was seen in all groups including controls. The severity of the lesions was greatest in the rats at in the high-exposure group.

The most significant gross observation in rats was the presence of enlarged bronchial and/or mediastinal lymph nodes, but these observations were not dose-related. Microscopically, this enlargement was attributable to an increase in normal constituents of the lymph nodes characterized by accumulations of macrophages, lymphocytes, neutrophils, and plasma cells. It was the opinion of the NTP Pathology Working Group (PWG) that hyperplasia of the lymph nodes and lower respiratory tract was typical of an infectious agent with an associated active immune response rather than ethyl benzene exposure. This diagnosis was supported by the following observations: an uneven distribution of lesions among and within groups; foci of airway inflammation were randomly distributed throughout the lungs; considerable variability in severity within groups; and there was no consistent concentration-response relationship. No lesions were seen in the nasal cavity. The PWG described these lesions as not typical of the type of lesions which occurs with known pulmonary irritants. These lesions were not found in control animals, which were housed in separate rooms. No infectious agent was identified upon serologic examination. In the draft NTP technical report (NTP, 1990), the inflammatory lung lesions were described as probably unrelated to exposure. Antibodies to common rodent respiratory tract viruses were not detected. However, only sera from control rats were sampled. Lesions morphologically indistinguishable from those in this study have been seen in control and treatment groups of rats from other inhalation and dosed feed studies. The PWG recommended that this effect be reevaluated in another study.

In mice, no significant exposure-related gross or histopathological observations were noted at terminal necropsy of any organs, including the lung. The only exposure-related effects were significantly elevated absolute and relative liver weight in both sexes of mice at of 750 and 1000 ppm and significantly elevated relative kidney weight of the females exposed to 1000 ppm. There were no significant histopathological changes or function test alterations in either liver or kidney of either sex.

Angerer and Wulf evaluated 35 workers who chronically (2-24 years, average 8.2 years) sprayed varnishes containing alkyd-phenol and polyester resins dissolved in solvent mixtures consisting principally of xylene isomers and ethyl benzene. Some of the varnishes contained lead-based pigments. The air samples from personal monitors indicated average levels of 4.0 ppm for ethyl benzene. Although workers had significantly elevated lymphocytes in addition to significantly decreased erythrocyte counts and hemoglobin levels compared with controls, these effects cannot be attributed to ethyl benzene since other compounds (e.g., xylene, methylchloroform, n-butanol, toluene, C9 hydrocarbons) were detected in some of the six workplaces evaluated.

Bardodej and Cirek carried out biomonitoring of 200 ethyl benzene production workers occupationally exposed for a mean duration of 12.2 years to unspecified concentrations of ethyl benzene and benzene over a 20-year period.

The workers were evaluated twice a year and ethyl benzene metabolites measured. No statistically significant differences in hematological effects (e.g., RBC, WBC, leukocyte and platelet counts) or liver function tests (e.g., aminotransferase and/or SAP and LDH activities and bilirubin tests) were observed between exposed and nonexposed workers.

NTP does not consider observations of lung lesions in rats exposed in the NTP subchronic study to be treatment-related. However, no infectious agent has been detected. Therefore, there remains a possibility that ethyl benzene may play a role in producing lung lesions. It is anticipated that this issue will be clarified upon completion of the chronic study in progress. In view of the previous considerations, the RfC is given a low confidence rating.

Ethyl benzene is classified a D carcinogen based on the lack of animal bioassays and human studies.

An HAONE of 3.2 x 10<sup>1</sup> mg/L and a NOAEL of 31.8 mg/kg/day were calculated with an uncertainty factor of 10 Appropriate data for calculating a Longer-term HA are not available. It is recommended that the DWEL of 3.4 mg/L be used as the Longer-term HA for the 70-kg adult.

Manganese. A oral RfD of 0.1 mg/kg/day and an NOAEL of 0.14 mg/kg/day with an uncertainty factor of 1 was reported. The information used to determine the oral RfD for manganese was taken from many large populations. Humans exert an efficient homeostatic control over manganese such that body burdens are kept constant with variations in diet. There are no subpopulations which are believed to be more sensitive to manganese at this level. The use of an uncertainty factor of 1 is supported by the fact that manganese is an essential element, being required for normal human growth and maintenance of health. It has also been suggested that children are less susceptible to manganese intoxication and may require slightly higher levels of manganese than do adults.

A small-scale epidemiologic study of manganese in drinking water was performed by Kondakis in three areas in northwest Greece. The mean concentration of manganese in hair was 3.51, 4.49, and 10.99 ug/g dry weight for areas A, B, and C, respectively (p<0.0001 for

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area C vs. A). However, the concentration of manganese in whole blood did not differ between the three areas.

A report by Kawamura described toxicologic responses in humans consuming large amounts of manganese dissolved in drinking water. The source of the manganese came from about 400 dry-cell batteries which were buried near a drinking water well. Sixteen cases of manganese poisoning were reported, with symptoms including lethargy, increased muscle tonus, tremor, and mental disturbances. The most severe symptoms were seen in elderly people, with children being affected to a lesser degree. Three individuals died, one from suicide. The cause of death for the other two was not reported, but the autopsy of one individual revealed manganese concentration in the liver to be 2 to 3 times higher than controls. Zinc levels were also increased in the liver. The well water was not analyzed until 1 month after the outbreak, at which time it contained approximately 14 mg Mn/L. However, when re-analyzed 1 month later, the levels were decreased by about half. Therefore, by retrospective extrapolation, the concentration of manganese at the time of exposure was probably at least 28 mg Mn/L. Assuming an adult body weight of 70 kg and a water consumption of 2 L/day, this would be equivalent to an intake of 0.8 mg Mn/kg bw/day from drinking water alone.

Rodents do not exhibit the same neurological deficits that humans do following exposure to manganese, so the relevance of these biochemical changes has been challenged. While primates are considered to be the species of choice for modeling the human response to manganese poisoning, only one limited oral study has been performed in a group of four rhesus monkeys. Muscular weakness and rigidity of the lower limbs developed after 18 months of exposure to 6.9 mg Mn/kg bw/day (as MnCl2-4H2O). Histological analysis showed degenerated neurons in the substantia nigra and scanty neuromelanin granules in some of the pigmented cells. An inhalation RfD of 4 x 10<sup>-4</sup> mg/cu.m and a LOAEL of 0.97 mg/cu.m with an uncertainty factor of 300. An uncertainty factor of 100 reflects 10 to protect sensitive individuals and 10 for use of a LOAEL. An additional factor of 3 was used to account for the less than chronic period of exposure.

Roels conducted a cross-sectional study in 141 male workers exposed to manganese dioxide, tetroxide and various salts (sulfate, carbonate and nitrate). A matched group of 104 male workers was selected as a control group. The two groups were matched for socioeconomic status and background environmental factors; in addition, both groups had comparable workload and workshift characteristics. The TWA of total airborne manganese dust ranged

from 0.07-8.61 mg/cu.m, respectively, with an overall mean and median of 1.33 and 0.97 mg/cu.m. The authors noted that there was an increase in production between 1965 (440 metric tons) and 1981 (22,000 metric tons) and presumably exposure with time. Thus exposure, particularly for individuals with long employment durations, may have been lower. The duration of employment ranged from 1-19 years with a mean of 7.1 years. The particle size and purity of the dust were not reported. Neurological examination, psychomotor tests (simple reaction time, short-term memory and hand tremor), lung function test (forced vital capacity, forced expiratory volume, peak expiratory flow rate and maximal expiratory flow rate at 50 and 75 percent of the FVC), blood and urine tests and a questionnaire were used to assess possible toxic effects of manganese exposure. The questionnaire was designed to detect CNS and respiratory symptoms.

Concentration-response relationships between length of exposure or urinary manganese levels and the prevalence of abnormal CNS findings were not observed. A significantly higher prevalence of coughs during the cold season, dyspnea during exercise and recent episodes of acute bronchitis were found in the exposed group. Lung ventilatory parameters were mildly altered in the exposed smokers. Significant alterations were found in simple reaction time (visual), audioverbal short-term memory test, eye-hand coordination, and hand steadiness test in the workers exposed to manganese. In general, this study is adequate to derive a risk assessment, however, certain limitations should be noted. One shortcoming is the lack of adjustment for age in the psychomotor measures. Age-standardization was used in the short-term memory task, but not in the measures of reaction time and tremor (hand steadiness and eye-hand coordination). However, since the mean age of the control group was higher than that of the exposed group, the likely effect of a lack of age adjustment is to underestimate the effect of manganese. Another limitation of the Roels study was the lack of correction for multiple tests. Differences between control and exposed groups on several neurobehavioral measures were assessed with simple t tests or chi-square tests. With alpha = 0.05, one in twenty such tests could be found statistically significant by chance alone. However, it appears that this percentage was well exceeded, e.g., 5 or 8 reaction time measures were significant and 7 of 11 short-term memory measures were significant. Thus, these flaws in the Roels study do not appear to compromise its utility for risk assessment purposes. Based upon the increased psychomotor disturbances, a LOAEL of 0.97 mg/cu.m was identified [where the LOAEL(HEC) = 0.34 mg/cu.m].

Chandra examined 60 welders from three separate plants exposed to manganese fumes. In plant 1, the workers complained of frequent occurrence of colds, cough and short hyperpyrex-

ia. The workers of all three plants often reported insomnia. No other subjective effects were reported by the workers in plants 2 and 3. No hematological alterations were observed in hemoglobin, RBC and WBC counts. Positive neurological signs (brisk, deep reflexes in the legs and/or arms) were observed in 25, 50 and 45 percent of workers in plants 1, 2 and 3, respectively. Tremors were also observed in one and four workers in plants 1 and 2, respectively. No positive neurological signs were observed in the control workers. Although significant effects are reported for "deep reflexes" and "tremors," it appears that these endpoints were assessed through a non-blind neurological examination. The findings of Chandra et al., may be viewed as supportive. Increased serum calcium levels and urinary manganese levels were also observed in the welders. The calculated LOAEL(HEC) from the mean exposure of plant 1 is 0.11 mg/cu.m.

Manganese toxicity can vary depending upon the route of exposure. When ingested, manganese is considered to be among the least toxic of the trace elements. In inhalation particle size will determine the site of deposition in the respiratory tract. Generally, in humans, fine mode particles (<2.5 microns) preferentially deposit in the pulmonary region and coarse mode particles (>2.5 microns) deposit in the tracheobronchial and extrathoracic regions. Those particles depositing in the extrathoracic and tracheobronchial regions are predominantly cleared by the mucociliary escalator into the gastrointestinal tract where absorption will be quite low (about 3 percent). For manganese, another possibility exists. A brief report suggested that another heavy metal, aluminum, was directly transported to the brain via nasal olfactory pathways (i.e., from extrathoracic deposition). One could speculate that this pathway may operate for manganese, raising additional difficulty in understanding target site dosage. Particles deposited in the pulmonary region will be cleared predominantly to the systemic compartment by absorption into the blood and lymph circulation. From all these factors, we assume 100 percent absorption of particles deposited in the pulmonary region, recognizing that this ignores other mechanisms that are likely to occur to some unquantified degree.

Chronic manganese poisoning in workers has been recognized since 1837. The primary effects associated with manganese toxicity from inhalation exposure in humans are signs and symptoms of CNS toxicity (manganism) and pneumonia. Manganism is believed to result from disturbances in the extrapyramidal motor system. Canavan reported the occurrence of diffuse cellular changes in the cerebral cortex and cerebellum, degeneration of nerve cells, satellitosis, and gliosis in the basal ganglia in a manganese miner. The observed CNS toxicity can be divided into two stages: the first is dominated by psychological disturbances

that subside if manganese exposure is terminated; the second is predominantly a neurological disturbance, which occurs with continued manganese exposure and is not reversible. Manganese neurotoxicity can involve psychiatric as well as neurobehavioral disturbances. In some cases these effects may be reversible; in others, the effects may persist even after termination of manganese exposure. Headache and somnolence followed by insomnia and fatigability are some of the earlier observed symptoms. If exposure is continued, speech and gait disturbances, tremor, mask-like face, postural instability, emotional instability and hallucinations may occur. Numerous investigators have reported CNS effects in workers exposed to manganese dust or fumes. Although there is an extensive database on CNS effects in workers, limitations in the studies preclude describing a quantitative dose/response relationship. Manganese concentrations are often presented as a broad range and particle size distribution and/or chemical characterization is not reported or adequately characterized. In addition, the occurrence of other chemicals at the factory is often not reported. Despite the limitations of these studies, they do provide information for identifying an effect level; psychological disturbances and/or neurological disturbances appear to be associated with long-term exposure to levels of manganese exceeding 0.25 mg/cu.m.

Workers exposed to manganese dust have a higher incidence of respiratory effects. An increased incidence of colds, bronchitis and pneumonia was reported in workers exposed to manganese dust and junior high school students living near a ferromanganese factory. As discussed in regard to the CNS toxicity, the study limitations preclude the establishment of a dose-response relationship. Similar respiratory effects were also observed in animals. Other effects observed in humans include hematological cardiovascular and reproductive effects. Workers employed in three different factories (30-35 workers/factory) and 30 matched controls were examined for neurological and psychological alterations. The mean concentrations of atmospheric manganese for the three plants were 1.0, 3.0, and 7.0 mg/cu.m. The specific manganese compound and other contaminants were not reported. An increased incidence of headache, involuntary movements, fatigue and exhaustion, sleep disturbances, sialorrhea, seborrhea, speech disturbances, gait disturbance, exaggerated reflexes, depression, hallucination, and prolonged reaction time were observed in workers exposed to manganese. The most common effects were headache, involuntary movements, fatigue, and exhaustion. The incidence of headaches; involuntary movements; disturbances in sleep, speech, and gait; and exaggerated reflexes were significantly increased with increasing duration of employment. Significant effects were observed in all three plants, thereby indicating the a LOAEL of 1.0 mg/cu.m in this study. Concentration-response relationships for the incidence of involuntary movements, speech disturbances, gait disturbances and hallucinations were observed. No

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correlation between air and blood manganese levels was observed. From these data a LOAEL(HEC) of 0.36 mg/cu.m was calculated.

An increased incidence of pneumonia was observed in men employed at a potassium permanganate manufacturing facility during an 8-year period (n=40-124) as compared with a control group of workers (n=>5000). The levels of manganese in the dust ranged from 0.7-38.3 mg/cu.m of which 43-54 percent, respectively, was manganese dioxide (0.3-21 mg MnO2/cu.m, 0.2-13.2 mg Mn/cu.m). Approximately 80 percent of the particles were <0.2 um and nearly all were <1 um. The other major compounds in the dust included calcium and potassium; barium (1 percent) and sodium (0.1 percent) were also detected in the dust. The levels of calcium and potassium in the dust were not reported. Trace amounts of silica, iron and lithium were also detected. The incidence of pneumonia in the workers was 26 per 1000, compared to an average of 0.73 per 1000 in the control group. All cases were diagnosed as lobar or bronchopneumonia. Workers also complained of bronchitis and nasal irritation. In a continuation of the Lloyd-Davies study, Lloyd-Davies and Harding reported the results of sputum and nasopharynx cultures for four men diagnosed as having lobar or bronchopneumonia. With the exception of one of these cases, Lloyd-Davies and Harding concluded that it was unlikely that bacterial infection played a primary role in producing the consolidation present in the lung and that manganese dust, without the presence of other factors, caused the observed pneumonitis. Based upon the range of exposure to manganese (0.2-13.2 mg/cu.m), a LOAEL(HEC) range of 0.07-4.7 mg/cu.m can be estimated.

Saric examined 369 workers in a ferroalloy plant. Workers in two other plants (electrode plant, n=190; aluminum rolling mill, n=204) served as controls. The ferroalloy plant workers were exposed to 0.3-20.41 mg/cu.m manganese; the manganese levels in the electrode plant and aluminum rolling mill were 0.002-0.03 mg/cu.m and 0.00005-0.00007 mg/cu.m, respectively. The workers were exposed to either manganese dust or fumes. The manganese compound or compounds that the workers were exposed to was not reported. A significant increase in the following subjective symptoms was observed in the ferroalloy plant workers: fatigue, bad mood, irritability and hand tremor. One or more sign(s) of neurological impairment (e.g., tremor, pathological reflexes) was observed in 16.8 and 5.8 percent of the workers in the ferroalloy plant and electrode plant, respectively. A significant decrease in systolic blood pressure without a change in diastolic blood pressure was also reported in the ferroalloy plant workers. Saric and Lucic-Palaic reported that in these groups of workers, manganese exposure and smoking might have a possible synergistic effect on the occurrence of respiratory symptoms.

Chronic manganese psychosis (16.6 percent), neuropsychiatric manifestations (22.2 percent), hemi-parkinsonism (2.7 percent) and choreoathetosis (2.7 percent) were observed in 36 workers employed in the dry battery industry. The workers were exposed to a dust containing 65-70 percent manganese dioxide (6.8-42.4 mg/cu.m). Contaminants in the dust included ammonium chloride, zinc oxide, graphite, acetylene black, ammonium hydroxide, cerium thorium nitrate, magnesium nitrate, and mercuric chloride. The particle size distribution was not reported. The psychological manifestations included headache, memory disturbances, sleep disturbances, uncontrollable laughter, sexual impotence or diminished libido, impulsive acts, uncontrollable weeping, irritability or depression, and hallucinations.

Smyth observed 71 workers exposed to manganese dust or fumes and 71 matched controls. The manganese levels in the fumes (primarily as manganese tetroxide) were 0.12-13.3 mg/cu.m and the majority of the particles were <2 microns in size. The manganese dust was mainly ferromanganese, with small amounts of manganosite (MnO), hausmannite (manganese tetroxide), and iron oxide. The manganese level in the dust ranged from 2.1-12.9 mg/cu.m.; 95 percent of the particles were <5 microns in size. Neurological examination of the workers revealed five workers with signs of CNS impairment. Three of these workers were exposed to manganese fumes and the other two to manganese dust. The five affected were exposed to the upper end of the exposure range. It is unclear if other workers exhibited signs of neurobehavioral problems.

The available evidence obtained from small laboratory animals indicates that rats may display some of the neurochemical changes associated with manganism in humans; however, they do not exhibit the wide range of behavioral manifestations described in primates. Manganese accumulation appears to be relatively high in pigmented substantia nigra tissues. Since the primate (but not rodent substantia nigra) shows pigmentation, there is some basis for assuming species differences in accumulation and toxicity of manganese. Because the deposition and retention in the respiratory tract is dependent on particle size, the particle size distribution of the atmospheric manganese is likely to play a role in respiratory tract damage. Particle size of the manganese dust was often not reported in the occupational studies; therefore, comparisons between human and experimental animal data are difficult. However, the experimental animal data support the findings in manganese workers that manganese exposure results in an increased incidence of pneumonia, pulmonary congestion, and pulmonary emphysema.

One of the primary effects of manganese exposure in humans is an increased prevalence of respiratory symptoms (pneumonia, bronchitis, colds, and coughs). Respiratory effects have also been reported in animals. It is unlikely that exposure to manganese is solely responsible for the increased prevalence of respiratory symptoms. Rather, manganese exposure probably increases susceptibility to infection. This is supported by several animal studies that have demonstrated immunotoxicity following exposure to manganese and Streptococci, Enterobacter or Klebsiella.

Male and female guinea pigs (sample size not reported) were exposed to 22 mg/cu.m manganese dioxide (13.9 mg Mn/cu.m) for 24 hours; 87 percent of the particles were <3 microns in size. Groups of guinea pigs were exposed to Enterobacter cloacae 1 day prior to manganese exposure, immediately before manganese exposure, or immediately after manganese exposure. The decrease in the clearance of manganese dioxide from the lungs, decrease in lung macrophages, and increase in the number of lung leukocytes observed in animals exposed to Enterobacter 1 day prior to manganese exposure were significant when compared to the manganese exposure-only group.

Inhalation RfD of confidence was medium in the principal study. The LOAEL for respiratory and CNS effects was supported by several other human studies. Manganese is classified as a class D carcinogen.

The Drinking Water Criteria Document for Manganese has received OHEA review.

Mercury. A risk assessment for this substance/agent is under review by an EPA work group.

Mercury is classified as a class D carcinogen. When 39 BD III and BD IV rats were injected i.p. over 2 weeks with 0.1 ml metallic mercury and observed for their lifetimes, sarcomas were seen only in those tissues that had been in direct contact with the metal. No concurrent controls were reported.

Mitsumori fed groups SPF ICR mice. One adenoma was detected among 37 controls surviving to week 53 or beyond, and no tumors were seen in either control or exposed females. The possible presence of tumors at other sites was not reported in this preliminary communication.

Methyl mercury hydroxide administered in the diet to Drosophila melanogaster at 5 mg/L induced chromosomal nondisjunction. Methyl and phenyl mercury produced small increases in the rate of point mutations.

The relevance of data from studies of organic mercury to the possible carcinogenicity of inorganic mercury is uncertain.

Methylene Chloride. An oral RfD of 6 x 10<sup>-2</sup> mg/kg/day, an NOAEL of 5.85 and 6.47 mg/kg/day (for males and females, respectively), and an LOAEL of 52.58 and 58.32 mg/kg/day (for males and females, respectively) with an uncertainty factor of 100. The 100-fold factor accounts for both the expected intra- and interspecies variability to the toxicity of this chemical in lieu of specific data.

The study is given a high confidence rating because a large number of animals of both sexes were tested in four dose groups, with a large number of controls. Many effects were monitored and a dose-related increase in severity was observed. The data base is rated medium to low because only a few studies support the NOAEL. Medium confidence in the RfD follows.

A risk assessment for the inhalation study of methylene chloride is under review by an EPA work group.

Methylene chloride is classified as a class B2 probable human carcinogen based in inadequate human data and sufficient evidence of carcinogenicity in animals; increased incidence of hepatocellular neoplasms and alveolar/bronchiolar neoplasms in male and female mice, and increased incidence of benign mammary tumors in both sexes of rats, salivary gland sarcomas in male rats and leukemia in female rats. This classification is supported by some positive genotoxicity data, although results in mammalian systems are generally negative.

Human carcinogenicity data is inadequate. Neither of two studies of chemical factory workers exposed to dichloromethane showed an excess of cancers. The Ott study was designed to examine cardiovascular effects, and consequently the study period was too short to allow for latency of site-specific cancers. In the Friedlander study, exposures were low, but the data provided some suggestion of an increased incidence of pancreatic tumors. This study was recently updated to include a larger cohort, followed through 1984, and an investigation of possible confounding factors. A nonsignificant excess in pancreatic cancer

deaths was observed, which was interpreted by EPA as neither clear evidence of carcinogenicity in humans, nor evidence of noncarcinogenicity. An update of the Ott et al. (1983) study, based on longer follow-up, indicated possible elevation of liver and biliary tract cancers (TSCA section 8(e) submission no. 8eHQ-0198-0772 FLWP et seq., 1989).

Dichloromethane administered in the drinking water induced a significant increase in combined hepatocellular carcinoma and neoplastic nodules in female F344 rats and a nonsignificant increase in combined hepatocellular carcinoma and neoplastic nodules in male B6C3F1 mice. Two inhalation studies with dichloromethane have shown an increased incidence of benign mammary tumors in both sexes of Sprague-Dawley and F344 rats. Male Sprague-Dawley rats had increased salivary gland sarcoma and female F344 rats had increased leukemia incidence. Both sexes of B6C3F1 mice developed liver and lung tumors after dichloromethane treatment. In a 2-year study by the National Coffee Association groups of 85 F344 rats received dichloromethane in the drinking water. In female rats the incidence of combined hepatocellular carcinoma and neoplastic nodules was statistically significantly increased in the 50 and 250 mg/kg dose groups when compared with matched controls. The incidence of hepatocellular carcinoma alone was not significantly increased. The combined incidence of hepatocellular carcinoma and neoplastic nodules in controls and the 4 dose groups was similar to that for historical controls. Male rats showed no increase in liver tumors.

In the same National Coffee Association study, B6C3F1 mice dichloromethane in drinking water. Male mice had an increased incidence of combined neoplastic nodules and hepatocellular carcinoma. The increase was not dose-related, but the pairwise comparisons for the two mid-dose groups were reported to be statistically significant.

The hepatocellular carcinoma incidence alone for male mice was not significantly elevated. Female mice did not have increased liver tumor incidence. The EPA regarded this study as suggestive but not conclusive evidence for carcinogenicity of dichloromethane. Inhalation exposure of Syrian hamsters to dichloromethane did not induce neoplasia. Sprague-Dawley rats were exposed under the same conditions. Female rats administered the highest dose experienced significantly reduced survival from 18-24 months. Female rats showed a dose-related increase in the average number of benign mammary tumors per rat, although the numbers of rats with tumors were not significantly increased. A similar response was observed in male rats, but to a lesser degree. In the male rats there was a statistically significantly positive trend in the incidence of sarcomas of the salivary gland; the incidence was significantly elevated at the high dose. There is a question as to whether these doses reached

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the MTD, particularly in the hamsters and the male rats. In another study (Dow Chemical Co.), Sprague-Dawley rats were exposed by inhalation to dichloromethane. No salivary tumors were observed, but there was an exposure-related increase in the total number of benign mammary tumors in female rats, although the increase was not statistically significant in any individual exposure group.

Groups F344/N rats and B6C3F1 mice were exposed to dichloromethane by inhalation. Survival of male rats was low; however, this apparently was not treatment-related. Survival was decreased in a treatment-related fashion for male and female mice and female rats. Mammary adenomas and fibroadenomas were significantly increased in male and female rats after survival adjustment, as were mononuclear cell leukemias in female rats. Among treated mice of both sexes there were significantly increased incidences of hepatocellular adenomas and carcinomas, and of alveolarbronchiolar adenomas and carcinomas, by life table tests. Adenomas and carcinomas were significantly increased alone as well as in combination. In addition, there were significant dose-related increases in the number of lung tumors per animal multiplicity in both sexes of mice.

Dichloromethane was mutagenic for Salmonella typhimurium with or without the addition of hepatic enzymes and produced mitotic recombination in yeast. Results in cultured mammalian cells have generally been negative, but dichloromethane has been shown to transform rat embryo cells and to enhance viral transformation of Syrian hamster embryo cells. Although chlorinated solvents have often been suspected of acting through a nongenotoxic mechanism of cell proliferation, found methylene chloride to be unable to induce hepatocellular division in mice.

An oral slope factor of  $7.5 \times 10^{-3}$  mg/kg/day was reported for methylene chloride.

**Methyl ethyl ketone (MEK).** A risk assessment for this substance/agent is under review by an EPA work group.

Methyl ethyl ketone is classified as a class D carcinogen based on no human carcinogenicity data and inadequate animal data.

Data for animal carcinogenicity is inadequate. No data were available to assess the carcinogenic potential of methyl ethyl ketone by the oral or inhalation routes. In a skin carcinogenesis study, two groups of 10 male C3H/He mice received dermal applications of 50 mg of a

solution containing 25 or 29 percent methyl ethyl ketone in 70 percent dodecylbenzene twice a week for 1 year. No skin tumors developed in the group of mice treated with 25 percent methyl ethyl ketone. After 27 weeks, a single skin tumor developed in 1 of 10 mice receiving 29 percent methyl ethyl ketone.

Methyl ethyl ketone was not mutagenic for Salmonella typhimurium strains TA98, TA100, TA1535, or TA1537 with or without rat hepatic homogenates. Methyl ethyl ketone induced aneuploidy in the diploid D61, M strain of Saccharomyces cerevisiae. Low levels of methyl ethyl ketone combined with low levels of nocodazole (another inducer of aneuploidy), also produced significantly elevated levels of aneuploidy in the system.

**2-Methylphenol and 4-Methylphenol.** An oral RfD of 5 x 10<sup>-2</sup> mg/kg/day, an NOAEL of 50 mg/kg/day, and an LOAEL of 150 mg/kg/day were determined with an uncertainty factor of 1000, 10 for interspecies and 10 for intraspecies variability and 10 for uncertainty in extrapolation of subchronic data to levels of chronic effects.

Thirty Sprague-Dawley rats were exposed to 2-methylphenol. The rats showed a high combined mortality, and a reduction in body weight. Food consumption was also significantly reduced. Kidney-to-body weight ratio was higher than that of the control value at the end of the study. In addition to the above effects, CNS effects such as lethargy, ataxia, coma, dyspnea, tremor, and convulsions were seen within 15 to 30 minutes after dosing; recovery occurred within 1 hour postgavage.

Another experiment with exposing Sprague-Dawley to 2-methylphenol monitored the following for signs of neurotoxicity: salivation, urination, tremor, piloerection, diarrhea, pupil size, pupil response, lacrimation, hypothermia, vocalization, exophthalmia, palpebral closure, convulsions (type and severity), respiration (rate and type), impaired gait, positional passivity, locomotor activity, stereotypy, startle response, righting reflex, performance on a wire maneuver, forelimb strength, positive geotrophism, extensor thrust, limb rotation, tail pinch reflex, toe pinch reflex, and hind limb splay were also evaluated. The lowest dose of o-cresol caused clinical signs of CNS-stimulation post dosing such a salivation, rapid respiration, and hypoactivity; however, these symptoms were low in incidence and sporadic in nature. Higher doses of o-cresol produced significant neurological events, such as increased salivation, urination, tremors, lacrimation, palpebral closure, and rapid respiration. High dosed animals also showed abnormal patterns in the neurobehavioral tests. The NOAEL based on systemic toxicity was 50 mg/kg/day in rats.

In a series of subchronic inhalation studies, Uzhdavine exposed rats and guinea pigs to o-cresol at a concentration of 9.0 (plus or minus 0.9) mg/cu.m. No effect was seen in guinea pigs. In rats, the authors reported various hematopoietic effects, respiratory tract irritation and sclerosis of lungs. Uzhdavine also reported that humans exposed to 6 mg/cu.m cresol (duration unspecified) experienced nasopharyngeal irritation. Other studies support the findings (effects) reported in this study. Based on a review and assessment of the available literature, primarily Uzhdavine, NIOSH recommended a TLV-TWA of 10 mg/cu.m (0.05 mg/kg/day). An RfD of 0.05 mg/kg/day can also be derived from this value; this lends support to the RfD derived from the subchronic toxicity studies.

Confidence is the study is high because the critical studies provided adequate toxicological endpoints that included both general toxicity and neurotoxicity. The data base is medium because there are adequate supporting subchronic studies. Thus, until additional chronic toxicity studies and reproductive studies are available, medium confidence in the RfD is recommended.

The health effects data for 2-methylphenol were reviewed by the U.S. EPA RfD/RfC Work Group and determined to be inadequate for the derivation of an inhalation RfC. The verification status for this chemical is currently not verifiable. 2-Methylphenol is classified as a class C carcinogen based on an increased incidence papillomas in mice in an initiation-promotion study. The three cresol isomers produced positive results in genetic toxicity studies both alone and in combination.

Only anecdotal data available is available for human carcinogenicity. Garrett reported two cases of multifocal transitional cell carcinoma of the bladder following chronic occupational exposure to cresol and creosote. Wodyka described a squamous cell carcinoma of the vocal cords in a petroleum refinery worker with a long history of exposure to cresol, dichloro-octane, and chromic acid.

Animal carcinogenicity is limited. Four skin application studies which had positive results are reported; however, the final two studies are of limited value due to the application of a mixture of chemicals. In a study by Boutwell and Bosch, female Sutter mice received a single dermal application of dimethylbenzanthracene (DMBA) in acetone as the initiator, followed 1 week later o-, m- or p-cresol in benzene twice weekly for 12 weeks. Skin papillomas were evaluated at 12 weeks. Many of the cresol-treated mice died, presumably of cresol toxicity. There was no mortality or evidence of skin papillomas in the benzene control

group (benzene weekly after DMBA initiation). None of the 12 mice in the benzene control group died or developed skin papillomas.

In another experiment, groups of 20 mice received a single dose DMBA in acetone, followed by twice weekly applications of m-cresol in benzene or p-cresol in benzene for 20 weeks. No skin papillomas were observed in the 18 surviving benzene control mice; m-cresol- and p-cresol-treated mice developed skin papillomas. These two experiments indicate that cresols can serve as tumor promoters of a polycyclic aromatic hydrocarbon.

Studies on the induction of unscheduled DNA synthesis showed p-cresol to be positive in human lung fibroblast cells in the presence of hepatic homogenates, the mixture of the three isomers to be weakly positive in primary rat hepatocytes, and o-cresol to be negative in rat hepatocytes.

In cell transformation assays using BALB/3T3 cells, a mixture of 3 cresol isomers was positive, and o-cresol was negative. Positive mutagenic responses were found at noncytotoxic doses. In another cell transformation assay using p-cresol, negative results were obtained with the mouse fibroblast cell line C3H1OT1/2.

Cresols (o-, m- and p-) are not mutagenic for various strains of Salmonella typhimurium both in the presence and absence of mammalian liver homogenates.

A mixture of the three isomers was mutagenic in a mouse lymphoma forward mutation assay with mammalian liver homogenates, while o-cresol was not mutagenic both with and without liver homogenates.

No isomer, when tested individually, induced sister chromatid exchanges (SCEs) in vivo, but the mixture of the three isomers induced SCEs in Chinese hamster ovary (CHO) cells in vitro. Only o-cresol induced SCEs in human lung fibroblasts and CHO cells.

In a screening test for putative carcinogens, infectious virus particles were produced from SV40-transformed weanling Syrian hamster kidney cells exposed to m-cresol.

o-Cresol is rated as a very toxic compound with a probable oral lethal dose in humans of 50-500 mg/kg, or between 1 teaspoon and 1 ounce for a 70 kg (150 lb.) person. o-Cresol is a strong dermal irritant and frequently causes dermatitis. Serious or fatal poisoning may result

if large areas of skin are wet with cresylic acid and the substance is not removed immediately. Ingestion of even a small amount may cause paralysis and coma. o-Cresol is corrosive to body tissues with toxicity similar to phenol.

Exposure to o-cresol may result in a burning pain in the mouth and throat; white necrotic lesions in the mouth, esophagus and stomach; abdominal pain, vomiting, diarrhea, paleness; sweating; weakness; headache; dizziness; ringing in ears; shallow respiration with "phenol" odor on the breath; scanty, dark-colored or "smoky" urine; and possibly delirium followed by unconsciousness. Convulsions are rarely seen, except in children. Hypersensitivity develops in certain individuals.

**Naphthalene.** A risk assessment for this substance/agent is under review by an U.S. EPA work group. No RfC is available at this time.

## CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Naphthalene is a D carcinogen, not classifiable as to naphthalene is a human carcinogenicity.

The National Toxicology Program is currently evaluating naphthalene for carcinogenicity in mice by the inhalation route; final results are not yet available. A group of 28 rats was exposed to naphthalene in the diet (6 times/week, average daily dose 30-60 mg/kg/day). No carcinogenic responses were reported. Mice were exposed to naphthalene via inhalation in a short-term pulmonary tumor bioassay. There was a statistically significant increase in the number of adenomas per mouse lung, but there was no apparent dose-response relationship. Tsuda et al. (1980) gavaged rats with naphthalene (single dose of 100 mg/kg) after a partial hepatectomy. At 2 weeks after surgery, 2-acetylaminofluorene (2AF) was added to the diet at 200 ppm, after 1 week of 2AF, a single 2.0 mL/kg dose of carbon tetrachloride was given. Feeding of 2AF continued for 1 week, followed by a basal diet for 1 week. Neither the number nor the size of gamma-glutamyl transpeptidase (GGT) foci appeared to be increased in naphthalene-treated rats compared with vehicle controls. A group of 10 rats received intraperitoneal injections of naphthalene (20 mg/rat) once a week for 40 weeks. No carcinogenic responses were reported. Coal tar-derived naphthalene with impurities was administered to rats subcutaneously (500 mg/kg) at 2-week intervals. Lymphosarcomas were found in 14.7 percent whereas controls had 2 percent incidence. This study is of limited value because of impurities and carbofuchsin was applied dermally to the injection site. Mice were painted with 0.5 percent coal tar-derived naphthalene with impurities in benzene 5 days/week for life. The value of this study for assessing carcinogenicity is very limited due to the presence of

potentially carcinogenic impurities and the vehicle is a known carcinogen. Other mouse skin-painting tests of naphthalene as a complete carcinogen and as an initiator of carcinogenicity were negative or inconclusive.

With one exception naphthalene was not positive when tested in a variety of genotoxicity assays. In the Ames test, naphthalene at concentrations of up to 2.5 mg/plate was not positive either with or without hepatic homogenates.

In a DNA damage assay, Nakamura reported that naphthalene was not positive. In phage induction assays, naphthalene did not yield positive results. DNA damage assays with naphthalene were not positive. Transformation assays were not positive.

**Nitrate.** An oral RfD of 1.6 mg/kg/day, an LOAEL of 1.8 to 3.2 mg/kg/day, and an NOAEL of 1.6 mg/kg/day were reported with an uncertainty factor of one. An uncertainty factor of 1 was employed because available data define the no-observed-adverse-effect level for the critical toxic effect in the most sensitive human subpopulation.

Most cases of infant methemoglobinemia are associated with exposure to nitrate in drinking water used to prepare infants' formula at levels >20 mg/L of nitrate-nitrogen. Cases reported at levels of 11-20 mg/L nitrate-nitrogen are usually associated with concomitant expostre to bacteriologically contaminated water or excess intake of nitrate from other sources.

Bosch evaluated 139 cases of cyanosis due to methemoglobinemia reported by physicians in Minnesota. All of the cases were in young children (ages 8 days to 5 months), with 90% occurring in infants <2 months of age. A study of the nitrate concentration of the wells used to supply water to the children with methemoglobinemia was performed. None of the wells contained <10 mg/L nitrate-nitrogen. Two wells contained 10-20 mg/L, although the diagnosis of methemoglobinemia was considered questionable in both these cases. There were 25 wells that contained 21-50 mg/L, 53 that contained 51-100 mg/L, and 49 that contained >100 mg/L nitrate-nitrogen. Nearly all the wells were shallow with inadequate protection from surface contamination. Coliform organisms were detected in 45 of 51 samples tested for bacterial contamination.

Walton described a survey performed by the American Public Health Association to identify clinical cases of infantile methemoglobinemia that were associated with ingestion of nitrate-contaminated water. A total of 278 cases of methemoglobinemia were reported. Of

214 cases for which data were available on nitrate levels in water, none occurred in infants consuming water containing <10 mg nitrate-nitrogen/L (1.6 mg nitrate-nitrogen/kg/day). There were 5 cases in infants exposed to 11-20 mg nitrate-nitrogen/L (1.8-3.2 mg/kg/day), 36 cases in infants exposed to 21-50 mg/L (3.4-8.0 mg/kg/day), and 173 in infants exposed to >50 mg/L (>8 mg/kg/day). Data on the ages of the infants were not provided.

Cornblath and Hartmann supplied nitrate-containing water to eight healthy infants (ages 2 days to 11 months) at doses of 50 or 100 mg NO3/kg/day (11 or 23 mgnitrate-nitrogen/kg/day). Assuming average consumption of about 0.16 L/kg/day, this corresponds to concentrations of 70 or 140 mg nitrate-nitrogen/L. No cyanosis was evident in any infant, and the highest concentration of methemoglobin was 7.5 percent. These authors also administered doses of 100 mg/kg of nitrate to four healthy infants (age 2 days to 6 months) and to two infants (age 6 and 7 weeks) who had been admitted to the hospital for cyanosis. No cyanosis was produced in the healthy infants, but cyanosis did occur in the individuals with a prior history of cyanosis. Examination of the saliva, gastric juice and stools of these infants revealed the presence of bacteria that readily reduced nitrate to nitrite. The gastric pH of these infants was >4 in both cases.

Simon measured methemoglobin levels in 89 healthy infants who received nitrate-free water, 38 infants who received water containing 11-23 mg nitrate-nitrogen/L (1.8-3.7 mg nitrate-nitrogen/kg/day), and 25 infants receiving water containing >23 mg nitrate-nitrogen/L (>3.7 mg nitrate-nitrogen/kg/day). For infants age 1-3 months, mean methemoglobin levels in these three groups were 1.0, 1.3 and 2.9 percent, respectively. For infants age 3-6 months, values were 0.8, 0.8 and 0.7 percent, respectively. No clinical signs of methemoglobinemia were detected in any of the infants.

Nitrate toxicity is due primarily to its conversion to nitrite, which oxidizes the Fe(+2) form of iron in hemoglobin to the Fe(+3) state. This compound (methemoglobin) does not bind oxygen, resulting in reduced oxygen transport from lungs to tissues. Low levels of methemoglobin occur in normal individuals, with typical values usually ranging from 0.5 to 2.0 percent. However, due to the large excess capacity of blood to carry oxygen, levels of methemoglobin up to around 10 percent are not associated with any significant clinical signs. Concentrations above 10 percent may cause a bluish color to skin and lips (cyanosis), while values above 25 percent lead to weakness, rapid pulse and tachypnea. Death may occur if methemoglobin values exceed 50-60 percent.

Conversion of nitrate to nitrite is mostly mediated by bacteria in the gastrointestinal system. Consequently, the risk of methemoglobinemia from ingestion of nitrate depends not only on the dose of nitrate, but also on the number and type of enteric bacteria.

The Food and Drug Administration sponsored extensive tests of the reproductive and developmental effects of NaNO3 and KNO3 in mice, rats, hamsters and rabbits. Fetuses were delivered by Cesarean section and examined for visceral and skeletal malformations. No significant effects were detected regarding maternal reproductive parameters (percent pregnant, abortion frequency, number of litters), fetotoxicity (percent fetal resportions, live fetuses per dam, average fetal weight) or fetal malformations up to the maximum doses administered to each species. These studies identify a reproductive/developmental NOAEL of 66 mg nitrate-nitrogen/kg/day for mice and hamsters and 41 mg nitrate-nitrogen/kg/day for rats and rabbits.

Sleight and Atallah studied the effects of nitrate on reproduction and development in guinea pigs. Normal conception occurred at all dose levels. No significant effect on reproductive performance was detected except in the high-dose group, where there was a decrease in number of live births. The authors attributed the fetotoxic effects to hypoxia due to maternal methemoglobinemia, although data on this were not provided. No fetal malformations were observed at any dose. This study identifies a reproductive NOAEL of 507 and a LOAEL of 1130 mg nitrate-nitrogen/kg/day.

Druckrey supplied rats with NaNO2 in drinking water for three generations. No teratogenic effects or adverse effects on reproduction were detected in any generation. Assuming that a maximum of 10 percent of a dose of nitrate is converted to nitrite by an adult human, this would correspond to a NOAEL of 200 mg nitrate-nitrogen/kg/day.

No studies were located on systemic effects of nitrate in humans or animals. In the absence of such data, observations from animals exposed to nitrite may be used as a conservative estimate of nitrate toxicity.

Shuval and Gruener exposed rats to water containing sodium nitrite. Histological examination of the lungs revealed dilated bronchi, fibrosis and emphysema at 1000 ppm or above. Histological examination of the heart revealed an increased percentage of coronary arteries that were characterized as "thin and dilated." This effect appears to be at least partly due to the absence of coronary artery thickening and narrowing that normally occurs in aged rats, so

it is not certain that these changes are inherently adverse. Based on effects on the lung, this study identifies a NOAEL of 2 and a LOAEL of 20 mg nitrite-nitrogen/kg/day. Assuming that a maximum of 10% of a dose of nitrate is converted to nitrite by an adult human, this would correspond to a NOAEL of 20 and a LOAEL of 200 mg nitrate-nitrogen/kg/day.

The studies of Bosch and Walton provide convincing evidence that infantile methemoglobinemia does not occur at drinking water levels of 10 mg nitrate-nitrogen/L or less. This is supported by a large number of additional epidemiological and case studies in humans (e.g., Cornblath and Hartmann; Simon; Toussaint and Selenka; Craun.

Polychlorinated Biphenyls (PCB). No assessment for noncarcinogen effects is available at this time.

Although there are many studies, the data are inadequate due to confounding exposures or lack of exposure quantification.

PCB mixtures assayed in the following animal studies were commercial preparations and may not be the same as mixtures of isomers found in the environment. Although animal feeding studies demonstrate the carcinogenicity of commercial PCB preparations, it is not known which of the PCB congeners in such preparations are responsible for these effects, or if decomposition products, contaminants or metabolites are involved in the toxic response. Numerous animal studies with PCBs have been conducted.

Most genotoxicity assays of PCBs have been negative. Peakall reported results indicative of a possible clastogenic action by PCBs in dove embryos.

The Oral Slope Factor is 7.7/mg/kg/day.

**Phenol- 108-95-2.** The oral RfD for phenol is  $6 \times 10^{-1}$  based on animal studies.

The evaluations of subchronic, chronic and reproductive/developmental studies indicated that phenol administered to pregnant rats at 120 mg/kg/day caused significant depression in fetal body weights, establishing this endpoint as the critical effect. Therefore, it is inappropriate to use NOAELs of 140 mg/kg/day for mice or 153 mg/kg/day for rats. The LOAEL for fetotoxicity was established at 120 mg/kg/day and the highest NOAEL at 60 mg/kg/day.

The health effects data for phenol have been reviewed by the U.S. EPA RfD/RfC Work Group and determined to be inadequate for derivation of an inhalation RfC.

Phenol is a Class D carcinogen based on no human carcinogenicity data and inadequate animal data. In carcinogenicity bioassays conducted by the National Cancer Institute concluded that, under these conditions, phenol was not carcinogenic in mice or rats.

Studies indicate that phenol may be a promoter and/or weak skin carcinogen in specially inbred sensitive mouse strains.

**Selenium and Compounds.** The oral RfD for selenium is 5 x 10<sup>-3</sup> mg/kg/day.

Although this is based on a human epidemiological study in which a sizable population with sensitive subpopulations was studied, there are still several possible interactions that were not fully accounted for, e.g., fluoride intake and protein status. Also, except for clinical signs of selenosis there are no other reliable indicators, biochemical or clinical, of selenium toxicity. Confidence in the data base is high because many animal studies and epidemiologic studies (reviewed by Combs) support the principal study. An additional human study with a freestanding NOAEL strongly corroborates the NOAEL identified in the principal study. Therefore, high confidence in the RfD is selected based upon support of the critical study and the high level of confidence in the data base.

Selenium is a Class D carcinogen based on inadequate human data and inadequate evidence of carcinogenicity in animals. The evidence for various selenium compounds in animals and mutagenicity studies is conflicting and difficult to interpret; however, evidence for selenium sulfide is sufficient for a B2 (probable human carcinogen) classification

Data on the potential carcinogenicity of selenium and various selenium compounds in humans are inadequate. Epidemiological studies have evaluated selenium in blood and cancer death rates in areas of high vs. low naturally-occurring selenium. However, these studies have limited value because they do not assess specific selenium compounds or correlate exposure with cancer risk.

**Silver.** An RfD of  $5 \times 10^{-3}$  mg/kg/day and an LOAEL of 0.014 mg/kg/day were reported with and uncertainty factor of 3. An uncertainty factor of 3 is applied to account for minimal effects in a subpopulation which has exhibited an increased propensity for the development of

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argyria. The critical effect observed is a cosmetic effect, with no associated adverse health effects. Also, the critical study reports on only 1 individual who developed argyria following an i.v. dose of 1 g silver (4 g silver arsphenamine). Other individuals did not respond until levels five times higher were administered. No uncertainty factor for less than chronic to chronic duration is needed because the dose has been apportioned over a lifetime of 70 years.

The critical effect in humans ingesting silver is argyria, a medically benign but permanent bluish-gray discoloration of the skin. Argyria results from the deposition of silver in the dermis and also from silver-induced production of melanin. Although silver has been shown to be uniformly deposited in exposed and unexposed areas, the increased pigmentation becomes more pronounced in areas exposed to sunlight due to photoactivated reduction of the metal. Although the deposition of silver is permanent, it is not associated with any adverse health effects. No pathologic changes or inflammatory reactions have been shown to result from silver deposition.

Silver compounds have been employed for medical uses for centuries. In the nineteenth and early twentieth centuries, silver arsphenamine was used in the treatment of syphillis; more recently it has been used as an astringent in topical preparations. While argyria occurred more commonly before the development of antibiotics, it is now a rare occurrence. Greene and Su have published a review of argyria.

Gaul and Staud reported 70 cases of generalized argyria following organic and colloidal silver medication, including 13 cases of generalized argyria following intravenous silver arsphenamine injection therapy and a biospectrometric analysis of 10 cases of generalized argyria classified according to the quantity of silver present. In the i.v. study, data were presented for 10 males (23-64 years old) and for two females (23 and 49 years old) who were administered 31-100 i.v. injections of silver arsphenamine (total dose was 4-20 g) over a 2- to 9.75-year period. Argyria developed after a total dose of 4, 7 or 8 g in some patients, while in others, argyria did not develop until after a total dose of 10, 15 or 20 g. In the biospectrometric analysis of skin biopsies from 10 cases of generalized argyria, the authors confirmed that the degree of the discoloration is directly dependent on the amount of silver present. The authors concluded that argyria may become clinically apparent after a total accumulated i.v. dose of approximately 8 g of silver arsphenamine.

Humans are exposed to small amounts of silver from dietary sources. The oral intake of silver from a typical diet has been estimated to range from 27-88 ug/day. Tipton estimated a

lesser intake of 10-20 ug/day in two subjects during a 30-day observation period. Over a lifetime, a small but measurable amount of silver is accumulated by individuals having no excessive exposure. Gaul and Staud estimated that a person aged 50 years would have an average retention of 0.23-0.48 g silver (equivalent to 1-2 g silver arsphenamine). Petering estimated a much lower body burden of 9 mg over a 50-year period based on estimated intake, absorption, and excretion values; however, it is not clear how the final estimate was calculated. Furchner studied the absorption and retention of ingested silver (as silver nitrate, amount not specified) in mice, rats, monkeys and dogs. In all four species, very little silver was absorbed from the GI tract. Cumulative excretion ranged from 90 to 99 percent on the second day after ingestion, with <1 percent of the dose being retained in <1 week in monkeys, rats and mice. Dogs had a slightly greater retention. The authors used the data from the dog to estimate how much silver ingested by a 70 kg human would be retained. An "equilibrium factor" of 4.4 percent was determined by integrating from zero to infinity a retention equation which assumes a triphasic elimination pattern for silver with the initial elimination of 90 percent coming from the dog data. The first elimination half-time of 0.5 days was used "arbitrarily"; subsequent half-times of 3.5 days and 41 days were taken from a metabolic study by Polachek. Furchner considered their calculated equilibrium factor of 4.4 percent to be a conservative estimate for the amount of silver which would be retained by a 70 kg human. This figure was rounded to 4 percent and was used in the dose conversion (i.v. dose converted to oral intake) for the calculation of the RfD.

Argyria, the critical effect upon which the RfD for silver is based, occurs at levels of exposure much lower than those levels associated with other effects of silver. Argyrosis, resulting from the deposition of silver in the eye, has also been documented, but generally involves the use of eye drops or make-up containing silver. Silver has been found to be deposited in the cornea and the anterior capsule of the lens. The same deposition pattern was seen in the eyes of male Wistar rats following administration of a 0.66 percent silver nitrate solution to the eyes for 45 days. No toxicological effects were reported.

Toxic effects of silver have been reported primarily for the cardiovascular and hepatic systems. Olcott administered 0.1 percent silver nitrate in drinking water to rats for 218 days. This exposure (about 89 mg/kg/day) resulted in a statistically significant increase in the incidence of ventricular hypertrophy. Upon autopsy, advanced pigmentation was observed in body organs, but the ventricular hypertrophy was not attributed to silver deposition.

Hepatic necrosis and ultrastructural changes of the liver have been induced by silver administration to vitamin E and/or selenium deficient rats.

Investigators have hypothesized that this toxicity is related to a silver-induced selenium deficiency that inhibits the synthesis of the seleno-enzyme glutathione peroxidase. In animals supplemented with selenium and/or vitamin E, exposures of silver as high as 140 mg/kg/day (100 mg Ag/L drinking water) were well-tolerated.

The critical human study rates a medium confidence. It is an old study which offers fairly specific information regarding the total dose of silver injected over a stated period of time. One shortcoming of the study is that only patients developing argyria are described; no information is presented on patients who received multiple injections of silver arsphenamine without developing argyria. Therefore, it is difficult to establish a NOAEL. Also, the individuals in the study were being treated for syphilis and may have been of compromised health.

Confidence in the data base is considered to be low because the studies used to support the RfD were not controlled studies. For clinical case studies of argyria it is especially difficult to determine the amount of silver that was ingested.

Confidence in the RfD can be considered low-to-medium because, while the critical effect has been demonstrated in humans following oral administration of silver, the quantitative risk estimate is based on a study utilizing intravenous administration and thus necessitates a dose conversion with inherent uncertainties.

Silver is classified as a class D carcinogen. In animals, local sarcomas have been induced after implantation of foils and discs of silver. However, the interpretation of these findings has been questioned due to the phenomenon of solid-state carcinogenesis in which even insoluble solids such as plastic have been shown to result in local fibrosarcomas.

No evidence of cancer in humans has been reported despite frequent therapeutic use of the compound over the years.

Animal carcinogenicity is inadequate. Local sarcomas have been induced after subcutaneous (s.c.) implantation of foils and discs of silver and other noble metals. Furst (1979, 1981), however, cited studies showing that even insoluble solids such as smooth ivory and plastic

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result in local fibrosarcomas and that tin when crumbled will not. He concluded that i.p. and s.c. implants are invalid as indicators of carcinogenicity because a phenomenon called solid-state carcinogenesis may complicate the interpretation of the cause of these tumors. It is difficult to interpret these implantation site tumors in laboratory animals in terms of exposure to humans via ingestion. Within these constraints there are two studies given below in which silver per se appeared to induce no carcinogenic response.

Schmahl and Steinhoff reported, in a study of silver and of gold, that colloidal silver injected both i.v. and s.c. into rats resulted in tumors in 8 of 26 rats which survived longer than 14 months. In 6 of the 8, the tumor was at the site of the s.c. injection. In about 700 untreated rats the rate of spontaneous tumor formation of any site was 1 to 3 percent. No vehicle control was reported.

Furst and Schlauder evaluated silver and gold for carcinogenicity in a study designed to avoid solid-state carcinogenesis. The authors mentioned the existence of spontaneous tumors in Fischer rats, but reported only injection site tumors. They concluded that finely divided silver powder injected i.m. does not induce cancer.

No evidence of the mutagenicity of silver was shown in two available studies. Demerec studied silver nitrate for the possible induction of back-mutations from streptomycin dependence to nondependence in Eschericha coli. Silver nitrate was considered nonmutagenic in this assay.

Nishioka screened silver chloride with other chemicals for mutagenic effects using a method called the rec-assay. Silver chloride was considered nonmutagenic in this assay.

**Styrene.** Styrene may change in the near future pending the outcome of a further review now being conducted by the Oral RfD Work Group.

An oral RfD of 0.2 mg/kg/day, an NOAEL of 200 mg/kg/day, and an LOAEL of 400 mg/kg/day were reported with an uncertainty factor of 1000. The uncertainty factor of 1000 reflects 10 for both intraspecies and interspecies variability to the toxicity of this chemical in lieu of specific data, and 10 for extrapolation of subchronic effects to chronic effects.

Four beagle dogs/sex were gavaged with doses of styrene in peanut oil. No adverse effects were observed for dogs administered styrene at 200 mg/kg/day. In the higher dose groups,

increased numbers of Heinz bodies in the RBCs, decreased packed cell volume, and sporadic decreases in hemoglobin and RBC counts were observed. In addition, increased iron deposits and elevated numbers of Heinz bodies were found in the livers. Marked individual variations in blood cell parameters were noted for animals at the same dose level. Other parameters examined were body weight, organ weights, urinalyses, and clinical chemistry. The NOAEL in this study is 200 mg/kg/day and the LOAEL is 400 mg/kg/day.

Long-term studies in rats and mice showed liver, kidney, and stomach lesions for rats and no significant effects for mice. Rats receiving a low average daily oral dose of styrene showed no adverse effects, while those receiving higher doses showed reduced growth and increased liver and kidney weights. Other subchronic rat feeding studies found LOAELs in the 350-500 mg/kg/day range and NOAELs in the range of 100-400 mg/kg/day.

The principal study is well done and the effect levels seem reasonable, but the small number of animals/sex/dose prevents a higher confidence than medium at this time. The data base offers strong support, but lacks a bona fide full-term chronic study; thus, it is also considered to have medium confidence.

A risk assessment for this substance/agent is under review by an EPA work group.

**Tetrachloroethylene** (*PCE*). An oral RfD of 0.001 mg/kg/day, an NOAEL of 14 mg/kg/day, and an LOAEL of 71 mg/kg/day with an uncertainty factor of 1000. The uncertainty factor of 1000 results from multiplying factors of 10 to account for intraspecies variability, interspecies variability and extrapolation of a subchronic effect level to its chronic equivalent.

Buben and O'Flaherty exposed Swiss-Cox mice to tetrachloroethylene in corn oil by gavage. Liver toxicity was evaluated by several parameters including liver weight/body weight ratio, hepatic triglyceride concentration, DNA content, histopathological evaluation, and serum enzyme levels. Increased liver triglycerides were first observed in mice treated with 100 mg/kg. Liver weight/body weight ratios were significantly higher than controls for animals treated with 100 mg/kg. At higher doses, hepatotoxic effects included decreased DNA content, increased SGPT, decreased levels of G6P and hepatocellular necrosis, degeneration and polyploidy.

A NOEL of 14 mg/kg/day was established in a second study, as well. Groups of 20 Sprague-Dawley rats of both sexes were administered doses of PCE in drinking water. Males in the high-dose group and females in the two highest groups exhibited depressed body weights. Equivocal evidence of hepatotoxicity (increased liver and kidney weight/body weight ratios) were also observed at the higher doses.

Other data support the findings of the principal studies. Exposure of mice and rats to tetrachloroethylene by gavage for 11 days caused hepatotoxicity (centrilobular swelling) at doses as low as 100 mg/kg/day in mice. Mice were more sensitive to the effects of tetrachloroethylene exposure than rats. Increased liver weight was observed in mice at 250 mg/kg, while rats did not exhibit these effects until doses of 1000 mg/kg/day were reached. Relative sensitivity to man cannot be readily established but the RfD of 1E-2 mg/kg/day is protective of the most mild effects observed in humans [diminished odor perception/modified Romberg test scores in volunteers exposed to 100 ppm for 7 hours; roughly equivalent to 20 mg/kg/day].

The principal studies are of short duration. Inhalation studies have been performed which indicate that the uncertainty factor of 10 is sufficient for extrapolation of the subchronic effect to its chronic equivalent. Liver enlargement and vacuolation of hepatocytes were found to be reversible lesions for mice exposed to low concentrations of tetrachloroethylene. In addition, elevated liver weight/body weight ratios observed in animals exposed to tetrachloroethylene for 30 days were similar to those in animals exposed for 120 days. Several chronic inhalation studies have also been performed. None are inconsistent with a NOAEL of 14 mg/kg/day for tetrachloroethylene observed by Buben and O'Flaherty and Hayes.

No one study combines the features desired for deriving an RfD: oral exposure, large number of animals, multiple dose groups, testing in both sexes and chronic exposure. Confidence in the principal studies is low mainly because of the lack of complete histopathological examination at the NOAEL in the mouse study. The data base is relatively complete but lacks studies of reproductive and teratology endpoints subsequent to oral exposure; thus, it receives a medium confidence rating.

Male Swiss-Cox mice were administered tetrachloroethylene by gavage weeks. Liver toxicity was evaluated by several parameters including liver weight-to-body weight ratio, hepatic triglyceride concentrations, DNA content, histopathological evaluation and serum enzyme levels. Increased liver triglycerides were first observed in mice treated with 100 mg/kg.

Liver weight/body weight ratios were significantly higher than controls for the 100 mg/kg group, and slightly higher than controls in the 20 mg/kg group. A NOAEL of 20 mg/kg/day was identified based on the absence of hepatotoxic effects. After 5 days of exposure, a NOAEL of 20 mg/kg/day was identified.

**Toluene.** The oral RfD for toluene is 2 x 10<sup>-1</sup> mg/kg/day based on studies with rats.

An uncertainty factor of 1000 was applied to account for inter- and intraspecies extrapolations, for subchronic-to-chronic extrapolation and for limited reproductive and developmental toxicity data.

Several subchronic and chronic inhalation studies have been performed on toluene but are not considered to be suitable for deriving an oral RfD.

Confidence in the principal study is high because a sufficient number of animals/sex were tested in each of six dose groups (including vehicle controls) and many parameters were studied. The same protocol was tested in both mice and rats, with rats being identified as the more sensitive species.

Toluene is a Class D carcinogen based on human data and inadequate animal data. Toluene did not produce positive results in the majority of genotoxic assays.

1,1,1-Trichloroethane. No reference dose is available for 1,1,1-Trichloroethane (1,1,1-TCA). A risk assessment for this substance/agent is under review by an U.S. EPA work group.

1,1,1-Trichloroethane is classified as a class D carcinogen. There are no reported human data and animal studies (one lifetime gavage, one intermediate-term inhalation) have not demonstrated carcinogenicity. Technical grade 1,1,1-trichloroethane has been shown to be weakly mutagenic, although the contaminant, 1,4-dioxane, a known animal carcinogen, may be responsible for this response.

The NCI treated Osborne-Mendel rats (50/sex/dose) with two concentrations of 1,1,1-trichloroethane 5 times/week for 78 weeks by gavage. Low survival of both male and female treated rats (3 percent) may have precluded detection of a significant number of tumors late in life. Although a variety of neoplasms was observed in both treated and

matched control rats, they were common to aged rats and were not dose-related. Similar results were obtained when the NCI treated B6C3F1 hybrid mice with the time-weighted average doses of 1,1,1-trichloroethane by gavage 5 days/week for 78 weeks. Survival was only 20 to 40 percent. A variety of neoplasms were observed in treated groups, but the incidence did not vary significantly from the controls.

Quast exposed 96 Sprague-Dawley rats of both sexes to 1,1,1-TCA vapor. There was an increase in incidence of focal hepatocellular alterations in female rats at the highest dosage.

The chemical failed to produce chromosomal aberrations in the bone marrow of cats, but responded positively in a cell transformation test with rat embryo cells.

An isomer, 1,1,2-trichloroethane, is carcinogenic in mice, inducing liver cancer and pheochromocytomas in both sexes.

A HAONE of 1 x  $10^2$  mg/L and a NOAEL of 1400 mg/kg/day have been reported with an uncertainty factor of 100 that allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study, and the assumption that a 10-kg child consumes 1L/day of water. Longer-term results report a HAONE OF 4 x  $10^1$  mg/L and a NOAEL of 350 mg/kg/day, also with an uncertainty factor of 100.

Rats were administered 1,1,1-trichloroethane by gavage. At levels above 0.5 g/kg reduced body weight gain and CNS effects were observed. Approximately 35 percent of these rats died during the first 50 days of the study. Also, the 5.0 g/kg/day dose group showed an increase in serum enzyme levels. The 0.5 g/kg/day level is identified as the NOAEL for this study. Based on a 7-day per week dosing regimen, this level would be equivalent to 350 mg/kg/day.

A longer-term (Adult) HA of  $1 \times 10^2$  mg/L and a NOAEL of 350 mg/kg/day with an uncertainty factor of 100 assuming a 70-kg adult consumes 2 L of water/day. In a study by Bruckner lifetime HA of  $2 \times 10^{-1}$  mg/L was reported.

McNutt continually exposed male mice to 1,1,1-trichloroethane via inhalation at various levels. Animals exposed to 5,460 mg/m<sup>3</sup> displayed significant changes in the centrilobular hepatocytes. Based on the conditions of exposure and an assumed absorption rate of 30 percent, the LOAEL of 1,365 mg/m<sup>3</sup> is equivalent to 35 mg/kg/day.

No information is available on the organoleptic properties of 1,1,1-trichloroethane. Treatment technologies which will remove 1,1,1-trichloroethane from water include granular activated carbon adsorption and boiling. Air stripping is also an effective method; however, this process transfers the contaminant directly to the air stream.

1,1,1-Trichloroethane is a list "C" Pesticide.

The final RQ is based on aquatic and chronic toxicity. Available data indicate a 96-hour Median Threshold Limit between 10 and 100 ppm, which corresponds to an RQ of 1000 pounds. RQ assignments based on chronic toxicity reflect two primary attributes, the minimum effect dose (MED) levels for chronic exposure (mg/day for 70-kg man) and the type of effect (teratogenicity, etc.). The composite score of these attributes for this substance is 6.0, corresponding to an RQ of 1000 pounds.

**Trichloroethylene.** A risk assessment for the oral and inhalation RfDs of this substance/agent is being evaluated by the EPA work group.

Trichloroethylene is classified as a class B2 carcinogen due to insufficient data for human carcinogenicity and sufficient data on animal carcinogenicity.

No experiments were reported for this compound.

**Vanadium.** A risk assessment for the oral RfD of this substance/agent is under review by an EPA work group.

No experiments were reported for vanadium.

Both chronic and subchronic oral RfDs for vanadium were reported as 7 x 10<sup>-3</sup> in the Health Effects Assessment Summary Tables (HEAST).

Vinyl Chloride. A risk assessment for the oral and inhalation RfDs of this substance/agent is under review by an EPA work group.

No experiments were reported for vinyl chloride.

An oral RfD of 1.9 mg/kg/day and an inhalation RfD of 8.4 x 10<sup>-5</sup> g/m <sup>3</sup> were reported in the Health Effects Assessment Summary Tables (HEAST). Vinyl chloride is listed as a class A human carcinogen.

**Xylenes.** The oral RfD is 2 mg/kg/day based on studies with rats.

Groups of rats were given gavage doses of 0, 250, or 500 mg/kg/day (rats) and 0, 500, or 1000 mg/kg/day (mice) for 5 days/week for 103 weeks. There was a dose-related increased mortality in male rats, and the increase was significantly greater in the high-dose group compared with controls. Although increased mortality was observed at 250 mg/kg/day, the increase was not significant. Mice given the high dose exhibited hyper-activity, a manifestation of CNS toxicity. There were no compound-related histopathologic lesions in any of the treated rats or mice.

Xylenes are Class D carcinogens based on a orally administered technical xylene mixtures did not result in significant increases in incidences in tumor responses in rats or mice of both sexes.

**Zinc and Compounds.** A risk assessment for this substance/agent is under review by an U.S. EPA work group.

Zinc is a Class D carcinogens based on inadequate evidence in humans and animals.

There are no reports on the possible carcinogenicity of zinc and compounds per se in humans. Case studies have been used to evaluate the effects of zinc administered for therapeutic reasons. There are reports which compare zinc levels in normal and cancerous tissue. Studies of occupational exposure to zinc compounds have also been conducted, but have limited value because they do not correlate exposure with cancer risk.

In a 1-year mouse study, zinc was administered in drinking water and the diet. None of these tumor incidences were significantly elevated in a statistical analysis of this data performed by the EPA. Guthrie performed a study of intratesticular injection of zinc. No conclusions about the carcinogenicity of the test zinc compounds could be made because an insufficient number of chickens were tested. Halme exposed tumor-resistant and tumor-susceptible strains of mice to zinc in drinking water. The tumor frequencies were higher than the spontaneous frequency, although no statistical analyses were reported.

## E.4.0 Toxicity Profiles for Essential Nutrients and Water Quality Parameters\_\_\_\_\_\_

**Calcium.** Acute single ingestions of calcium salts may product mild gastrointestinal distress; calcium chloride is more irritating and may cause hemorrhage. Hypercalcemia or other toxicity has not been reported from acute ingestion. Chronic ingestion of calcium carbonate may cause hypercalcemia, alkalosis, and renal impairment.

Chronic administration of 4 to 60 grams per day of calcium carbonate for 2 to 60 days has produced the "milk-alkali" syndrome. Chronic renal failure patients on hemodialysis may develop hypercalcemia with lower amounts.

No toxicity values (e.g., RfD, MCL) are available for calcium.

*Iron.* Exposure to iron may result in vomiting, diarrhea, minor lethargy, and hyperglycemia. More severe exposure may result in stupor, shock, acidosis, hematemesis, bloody diarrhea, or coma. No toxicity values are available for iron.

**Magnesium.** Hypermagnesiumemia leads to hypotension, ECG changes, and impairment of neuromuscular transmission. Magnesium dust can irritate the eyes and mucous membranes of the upper respiratory tract causing atrophic nasopharyngitis.

Insufficient data exists in the literature to assess the range of toxicity following acute overdose. In hypomagnesiumemia, 2 to 4 g/day may be tolerated orally.

No toxicity values are available for magnesium.

**Potassium.** Toxicity from oral ingestion of potassium salts is slight because potassium is readily excreted in the urine. In overdose situations, symptoms may range from vomiting and diarrhea, through listlessness and muscular cramps, to hypotension and arrhythmias.

No toxicity values are available for potassium.

**Sodium.** Death has followed the use of sodium chloride as an emetic. Symptoms may include vomiting, diarrhea, restlessness, thirst, dizziness, headache, convulsions, coma, tachycardia, hypotension, and respiratory arrest.

One level tablespoon (17.85 grams) is approximately 305 mEq of sodium, and if retained, would raise serum levels by 30.5 mEq/L in a 3-year-old child (estimating 10 L of extracellular fluid volume).

Central nervous system symptoms are common above 150 to 160 mEq/L. At these levels there is a 10 percent chance of convulsions which increases when levels reach 160 to 185 mEq/L. Death is a frequent occurrence above 185 mEq/L. An ingestion of 0.5 to 1 g/kg will be toxic in most patients.

No toxicity values are available for sodium.

Sodium Bicarbonate. Exposure to sodium bicarbonate may result in metabolic abnormalities, hypernatremia, hypokalemia, hypochloremia, alkalosis, and hypocalcemia. Dizziness, weakness, irritability, and mental status changes may be initial symptoms of alkalosis or hypernatremia. Progressive obtundation, coma, and seizures may occur in severe toxicity. Alkalosis has occurred after one tablespoonful in a young infant. Much larger amounts are needed to cause hypernatremia (10 to 20 g/kg). Adults with normal renal function can tolerate up to 1,700 mEq daily with minimal symptoms (HSDB, 1992).

No toxicity values (e.g., RfD) are available for bicarbonate.

**Sodium Chloride.** An ingestion of 0.5 to 1 g/kg can be toxic to most patients. Gastric mucosal irritation with vomiting and gastrointestinal ulceration (gastric mucosa, esophagus, duodenum) has been reported. Initially, the skin loses its turgor, the mucous membranes become dry, and the muscles weaken; there is increased thirst and decreased appetite. With dehydration comes a rise in body temperature and a metabolic acidosis. In babies, the anterior fontanelle becomes flat. The brain volume decreases rapidly, leading initially to an increase and then a decrease in deep tendon reflexes, muscle rigidity, an increase in irritability, lethargy, opisthotonos, seizures, coma, and death resulting from circulatory collapse or CNS damage. If the patient survives, there may be residual neurologic damage (HSDB, 1992).

No toxicity values (e.g., RfD) are available for chloride.

**Sodium Fluoride.** Following ingestion, sodium fluoride probably reacts with gastric acid to produce highly corrosive HF that causes nausea, vomiting, diarrhea, abdominal pains, and acute hemorrhagic gastroenteritis following massive overdose.

In most instances, gastrointestinal signs and symptoms predominate the clinical scene. The minimum toxic or lethal dose is not well established in the literature and wide variations in the response to a given dose among different individuals are noted.

Accidental ingestion of sodium fluoride by children usually does not present serious risk if the amount of fluoride ingested is less than 5 mg/kg.

Prenatal fluoride supplementation (2.2 mg NaF or 1 mg fluoride daily) during the last two trimesters of pregnancy has been reported to be safe (HSDB, 1992).

No toxicity values (e.g., RfD) are available for fluoride.

**Sodium Sulfide.** Saline cathartics, such as sodium sulfate, are poorly absorbed from the gastrointestinal tract hence, systemic toxicity is unlikely unless massive amounts have been ingested. Nausea, vomiting, abdominal pain, and diarrhea are frequent findings. Severe diarrhea may result in excessive fluid and electrolyte loss (HSDB, 1992).

A minimal lethal or toxic dose has not been established in the literature and no toxicity values (e.g., RfD) are available for sulfate.

# VOLUME 4, APPENDIX F WEST SIDE AQUIFER TEST ANALYSIS

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#### F.1.0 Introduction

As part of the remedial investigation (RI) at Naval Air Station (NAS) Moffett Field (Moffett Field), hydraulic properties of subsurface water-bearing materials were determined through analysis of pump test data collected at two sites within Operable Unit 4 (OU4). These sites were:

- Site 8 Waste Oil Transfer Area
- Site 9 Old Fuel Farm and Old NEX Gas Station.

This report addresses pump testing conducted at Sites 8 and 9. Chapter F.1.0 presents the scope and objectives of the OU4 pump tests. Chapter F.2.0 describes the analysis methods and provides an overview of the theoretical basis for the methods used. Chapter F.3.0 provides a description of the general field approach. Results of the pump test analysis are presented in Chapter F.4.0, which is organized by site.

Also presented in this appendix are eight attachments. Attachments I through V present the water level field data collected during each pumping and recovery test. The data contained in these attachments are as follows:

- Attachment I Pump Test 7, Site 8
- Attachment II Pump Test 1, Site 9
- Attachment III Pump Test 3, Site 9
- Attachment IV Pump Test 5, Site 9
- Attachment V Pump Test 8, Site 9.

Attachment VI contains boring logs for the piezometers and pumping wells installed during the investigation. Attachment VII contains soil consolidation results, and Attachment VIII contains groundwater analytical results.

A single test (Pump Test 7) was conducted at Site 8. Four tests (Pump Tests 1, 3, 5, and 8) were conducted at Site 9. The locations of Sites 8 and 9 and their associated pump tests are shown in Figure F1-1. Pump Tests 5 and 8 included separate pumping sessions at two wells per test. The pump test numbering sequence is based on the order in which the tests are presented in the "Final Aquifer Test Plan" (IT, 1991a). A generalized cross section showing the screened intervals for observation points (monitoring wells and new piezometers) and the

pumping wells is provided in Figure F1-2. In Figure F1-2, two wells labeled PT9-1 and PT9-2 are large-diameter wells installed during the field investigation proceeding the aquifer test.

Pump Tests 2, 4, and 6 were conducted as part of Operable Unit 5 (OU5) activities and will be addressed in a separate report. These sites are:

- Site 4 Former Wastewater Holding Ponds
- Site 5 Old Fuel Farm Area.

**Scope and Objectives.** The pump tests addressed in this report were conducted in the A1-and A2-aquifer zones. These zones comprise the most shallow saturated materials present at Moffett Field. Groundwater contamination in the western portion of Moffett Field (OU4) is mainly restricted to the A1- and A2-aquifer zones.

Pump testing was conducted to provide quantitative estimates of the hydraulic properties of the A1- and A2-aquifer zones and of the intervening A1/A2 aquitard. These properties include transmissivity, hydraulic conductivity, and storativity. In addition, the pump tests were conducted to:

- Establish the extent of hydraulic interconnection within the zones of the A1- and A2-aquifer zones
- Provide additional characterization of lateral and vertical groundwater flow paths
- Provide data concerning the presence of recharge zones and impermeable boundaries
- Classify the hydraulic conditions within the A1- and A2-aquifer zones as confined, semiconfined, or unconfined.

These objectives were chosen to provide data needed for the baseline risk assessment, fate and transport modeling of groundwater contaminants, and for use in evaluating remedial alternatives.

#### F.2.0 Analytical Methods

Pump test data are analyzed to obtain estimates of hydraulic parameters and to establish the presence of recharge and impermeable boundaries. Analyses are made by plotting drawdown versus time on log-log or semilog paper and applying either graphical curve matching or mathematical treatment to the data. Several methods are applicable to pump test data depending on whether the aquifer is confined, unconfined, fractured, or may receive leakage from another aquifer.

Data analysis presented in this appendix was performed using AQTESOLV (Geraghty & Miller, 1989). AQTESOLV is an interactive computer software package capable of implementing a variety of aquifer models through a nonlinear least squares technique in which hydraulic parameters are estimated from observed data. Alternatively, the observed data can be graphically matched to theoretical-type curves. AQTESOLV incorporates analytical solutions for unsteady flow to a well using the methods developed by Theis (1935), Cooper-Jacob (1946), Hantush-Jacob (1955), Hantush (1960), and Cooper, et al. (1967). Aquifer parameter estimations are performed numerically by utilization of the Gauss-Newton linearization method. This method minimizes errors between observed and estimated values by truncating the Taylor series after the first differential form and adding a Marquardt correction factor where the Gauss-Newton method fails to converge.

Portions of the aquitard analysis were performed using techniques described by Neuman and Witherspoon (1969).

Drawdowns in several monitoring wells and piezometers were analyzed using a method for unconfined aquifers (Neuman, 1975). As will be discussed in Chapter F.4.0, the data were not well represented by the Neuman model. Therefore, background and results from the Neuman method are not included in this report.

The Theis (1935) equation and the Cooper-Jacob (1946) approximation to the Theis equation represent unsteady radial flow to a well in a confined aquifer. The following conditions are assumed:

- Aquifer is of infinite areal extent.
- Aquifer is homogeneous, isotropic, and of uniform thickness.
- Potentiometric surface is initially horizontal.

- Pumping rate is constant.
- · Pumping well is fully penetrating.
- · Water is released instantaneously from storage with decline in hydraulic head.
- Well storage is negligible.

Storage calculated from the Theis, Jacob-Hantush, Hantush, Cooper et al., and Cooper-Jacob solutions do not apply under conditions where delayed yield occurs, and may underestimate the actual yield during gravity drainage. However, the storage coefficients obtained are valid for the portions of the response curve during which the behavior follows the type curve. Pump tests were conducted over approximately a 24-hour period and, as discussed in Chapter F.4.0, the Theis solution did not account for delayed yield. As shown in Chapter F.4.0, all of the aquifer zones are classified as semiconfined with storage coefficients on the order of 7.7 x 10<sup>-5</sup> to 2.35 x 10<sup>-3</sup>. Unconfined aquifers typically exhibit storage coefficients of 0.1 to 0.001 (Freeze and Cherry, 1979). Data interpretation indicated that for several of the pump tests, the duration may have not been sufficient for the drawdown response curve to return to theoretically ideal behavior. Therefore, the storage values reported in this document may not take into account all delay yield effects.

The Cooper-Jacob approximation method is considered valid for well function arguments less than 0.01. This condition is met when the separation between pumping and monitoring wells is small and for extended pumping times.

The Hantush-Jacob method for unsteady flow to a well in a semiconfined aquifer incorporates the assumptions of the Theis equation with the following modifications:

- Aquitards are of infinite areal extent with uniform thickness and vertical hydraulic conductivity.
- Flow in the aguitards is vertical.
- Aquitards are bounded by infinite constant head boundaries.
- Aquitard storativity is zero.

Hantush and Jacob developed a dimensionless variable, r/B, to quantify relative leakage from an aquitard. The term r/B is part of the well function (Walton, 1960) for wells screened in leaky aquifers and is related to the radial distance from the pumped well (r), the thickness of the aquifer and aquitard, and the hydraulic conductivities of the aquitard and aquifer. If the aquitard is impermeable, the Hantush-Jacob solution reduces to the Theis solution when r/B

equals zero. These assumptions are also applied in the Hantush method for semiconfined aquifers with the exception that aquitard storage is assumed homogeneous and nonzero.

Pump test data obtained from monitoring wells and piezometers were evaluated using one of the aforementioned techniques. Observed drawdown data versus time were plotted on log-log scale and examined for deviation from the master Theis curve. Deviation from the theoretical curve was evaluated to determine the choice of an appropriate model. Flattening of the response curve indicated recharge, and a leaky model was applied. Over steepening of the response curve indicated diminishing yield and suggested either an impermeable boundary or lateral thinning of the aquifer unit. In some cases, data could not be matched to model generated curves. In these instances, deviations from the Theis curve were explained in terms of recharge boundaries, changes in aquifer geometry, or variation of hydraulic properties encountered by the expanding cone of depression.

Data from pumped wells were analyzed using the Theis recovery method. This technique, which has the same theoretical basis as the Theis method for confined aquifers, cannot be used to estimate storativity. Transmissivities estimated using the Theis recovery technique are considered less representative of actual aquifer conditions due to the restricted nature of the model and possible nonlinear flow conditions near the pumped well. Therefore, transmissivities estimated from recovery data validate the approximate magnitude of estimates considered to be more representative of actual aquifer conditions.

Slug test analyses were performed on data obtained from piezometers screened in the A1/A2 aquitard using the method of Cooper, et al. (1967), which incorporates assumptions similar to those of the Theis equation. The slug test analyses incorporate the assumption of a single instantaneous change in water level in the test piezometer.

Neuman and Witherspoon's ratio method of aquitard analysis was used to calculate vertical hydraulic conductivity for the A1/A2 aquitard. Values of specific storage (S<sub>e</sub>) were calculated from consolidation test data (American Society for Testing and Materials [ASTM] D2435). Specific storage is defined as the volume of water released per unit volume of aquifer under a unit decline in hydraulic head (Freeze and Cherry, 1979). Piezometers were placed in the aquitard at four locations to determine these values; however, the test procedures were valid for only two of the piezometers. Time-drawdown responses from two of the four piezometers indicated an aquitard connection (transmissive pathway) to the pumped aquifer and were

excluded from the analysis. The analyses of aquitard piezometers are included in the sections on aquifer analysis for each test site (Sections F.4.2.6 and F.4.3.6).

Compressibility ( $\alpha$ ) was determined by applying the following relationship (Kruesman and de Ridder, 1989):

$$\alpha = \{-de/(1+e_0)\}/d\sigma$$

where:

e = void ratio $\sigma = effective stress.$ 

The value of compressibility for the sample (classified as silty clay) was  $2.028 \times 10^{-7} \text{ m}^2/\text{N}$ . This value was applied in an equation for specific storage ( $S_s$ ) given as:

$$S_s = \rho g(\alpha + n\beta)$$

where:

 $\rho g = unit weight of water$ 

n = porosity (e/1+e)

 $\beta$  = compressibility of water.

Specific storage is defined as the volume of water released per unit volume of aquifer under a unit decline in hydraulic head (Freeze and Cherry, 1979). The calculated value of the aquitard specific storage is 0.0020 ft<sup>-1</sup>. This value was used in Neuman and Witherspoon's ratio method of aquitard analysis to arrive at a value of vertical hydraulic conductivity.

#### F.3.0 Field Methods

The pump tests at Moffett Field were conducted to determine aquifer characteristics in the areas of highest groundwater contamination -- the A1- and A2-aquifer zones. The pump test locations were chosen based on the concentrations of contaminants in monitoring wells and the estimated ability of targeted aquifer zones to withstand prolonged pumping.

At each site, the following procedure was followed:

- A step-drawdown test was conducted to obtain an optimum discharge rate.
- The pumping well and piezometers were allowed to recover after the stepdrawdown test, and then a 24-hour constant discharge pump test was conducted.
- Pressure transducers were placed in monitoring wells.
- Periodic measurements were also taken by hand at 7 to 21 monitoring wells during the test.
- Discharge rate was monitored constantly until the rate stabilized and every 30 to 60 minutes thereafter.
- At least one monitoring well outside of the expected zone of influence was monitored to observe changes in pump test site-wide water level changes.
- After the constant discharge test, selected wells were monitored for recovery over an 8-hour period.

Slug tests were performed on three A1- and A2-aquitard piezometers to determine the lateral transmissivity and storativity of the aquitard. Vertical hydraulic conductivity and specific storage were determined at two locations in the A1/A2 aquitard.

#### F.3.1 Well Placement

Piezometers were installed at predetermined locations to monitor hydrologic responses near the pumped well. Often, the piezometers were placed in clusters to monitor separate zones. Within the context of this report, piezometers will refer to the 2-inch-diameter wells that were installed to fully penetrate the aquifer of interest or to monitor a 1-foot-thick aquitard zone. Existing RI investigation monitoring wells located in the vicinity of pump tests were utilized as monitoring wells whenever possible.

The borehole for the deepest piezometer at each cluster was logged through visual classification of core samples to provide stratigraphic and lithologic information representative of the geologic conditions. Boring logs for newly installed piezometers and existing monitoring wells used in this investigation are included in Attachment VI. One piezometer at each cluster was screened in the same interval as the pumped well, unless that interval was not water-bearing (e.g., silt or clay). In this case, the water-bearing unit in closest vertical proximity to the pumped unit was screened. The aquitard piezometers screened a 1-foot-thick zone within the A1/A2 aquitard.

Following Lohman (1972), monitoring wells and piezometers were selected at radial distances of 1.5, 2.5, and 4 times the aquifer thickness from the pumping well where possible. Thicknesses of the A1- and A2-aquifer zones ranged between 3 and 20 feet at Sites 8 and 9. Piezometers installed in the A1/A2 aquitard were placed in the cluster nearest the pumping well in an effort to maximize response to pumping in the aquitard.

#### F.3.2 Equipment

The pumping system for both the step-drawdown tests and the constant discharge tests consisted of a submersible pump connected to a riser pipe. The outflow was fitted with an in-line totalizing flowmeter, a flow adjustment valve, flexible hose, and a 2,000-pound granular activated carbon (GAC) unit. The GAC unit was used to filter dissolved organic compounds from the discharged water. The discharge from the GAC unit was placed in a water containment tank. For the step-drawdown tests, a 4,000-gallon rolling Baker tank was used to store the water after it was filtered through the GAC unit. When full, the rolling tank was then moved to a larger 21,000-gallon tank and the water transferred. During the constant discharge tests, the 21,000-gallon tanks were placed near the pumping wells, and the water was directly discharged to the tanks after passing through the GAC unit.

Two submersible pumps were used. A Grundfos<sup>®</sup> 1-horsepower (hp) submersible pump was used for Pump Tests 3, 5 (A2), and 8 (A1). For Pump Tests 1, 5 (A1), 7, and 8 (A2), a Grundfos 2-hp pump was employed. Power was provided by a 5.5 kilowatt (kW) generator for the smaller pump, and a 10 kW generator for the larger pump.

An eight-channel data logger (Hermit® 2000) was used in conjunction with pressure transducers to record water level changes in monitoring wells and piezometers located near the pumping well. Readings were recorded at logarithmically-spaced time intervals during the

pumping and recovery periods. Water level meters were used to monitor the water levels in the remaining selected monitoring wells.

#### F.3.3 Groundwater Sampling

Groundwater samples were collected from the pumped well at approximately 8-hour intervals during each test. The samples collected were analyzed for volatile organic compounds (VOC) using U.S. Environmental Protection Agency (U.S. EPA) Method 8240 for purgeable organic components. In addition, temperature, pH, and specific conductivity measurements were obtained every 2 hours from the pumping well.

A field quality assurance (QA) sampling scheme was established to check sampling and analytical accuracy and precision. Field QA samples included the following:

- Duplicates
- Matrix spikes
- · Matrix spike duplicates
- Field blanks
- · Trip blanks.

All groundwater samples from the pumping wells were collected and controlled as defined in Section 5.7 (Groundwater Monitoring) of the Moffett Field Sampling and Analysis Plan (IT, 1988).

#### F.3.4 Disposal of Groundwater

Approximately 294,000 gallons of water generated during the aquifer test was contained at each site in 21,000-gallon tanks. Each tank was sampled and analyzed for VOCs to be certain that the on-site treatment by carbon adsorption had effectively removed the contaminants to a level below the maximum contaminant level (MCL). Results of sample analyses indicated that the carbon unit was effective. Of the 15 Baker tanks on location, only three had analysis results at or slightly above the detection limit of 0.5 parts per billion (ppb). All detections were of trichloroethene (TCE), and all were well below 5 ppb, which is the MCL for this chemical. The treated water will be disposed of by Moffett Field.

#### F.4.0 Pump Tests

Seven aquifer tests were conducted at five test sites to evaluate the hydraulic properties at those locations. At Site 8, the Waste Oil Transfer Area, one pump test was conducted (Pump Test 7). At Site 9, the Old Fuel Farm, six aquifer tests were performed (Pump Tests 1, 3, 5 [A1 and A2], and 8 [A1 and A2]). Pump Tests 1 and 3 targeted the A2-aquifer zone. For Pump Tests 5 and 8, pumping was independently conducted on both the A1- and A2-aquifer zones. Table F4.0-1 gives the drawdown in monitored wells and piezometers at the end of the pumping periods for each test.

#### F.4.1 Pump Test 7, Site 8

Pump Test 7 was conducted at Site 8 (Figure F4.1-1), which exhibits locally elevated concentrations of chlorinated hydrocarbons including trichloroethane (TCA) and TCE. Hydrogeological characterization of the aquifer units is required to provide data that will be necessary if a groundwater extraction and treatment system is required at this site. Figures F4.1-2 and F4.1-3 are geological cross sections of Site 8 based on the boring logs for monitoring wells installed during the RI field investigation, and on piezometers installed for this investigation. As shown in the cross sections, the A1- and A2-aquifer zones interfinger at Site 8. Well W08-06 is screened in a thick sand sequence, which appears to intersect portions of both the A1- and A2-aquifer zones and was therefore chosen as the pumping well for Pump Test 7. This well is located close to the center of Site 8. The monitoring wells for Pump Test 7 are listed in Table F4.1-1. Static water levels are above the upper confining unit or are in sand units, which are higher in elevation than the normal position of the A1-aquifer zone, thus the aquifer system here is either confined or semiconfined.

#### F.4.1.1 New Wells/Piezometers Installed

Two piezometers were installed for Pump Test 7 to provide drawdown data in the A1-aquifer zone (Table F4.1-2). The two piezometers installed for Pump Test 7, PZ8.7-1(A1) and PZ8.7-2(A1), were placed to fully penetrate the merged A1- and A2-aquifer zones encountered.

#### F.4.1.2 Site-Specific Geology

The A1- and A2-aquifer zones are characterized by predominantly fine-grained materials with interbedded sand and gravel deposits. Based on lithologic data collected during the monitoring well and piezometer installation, the A1- and A2-aquifer zones cannot be distinguished

and form a continuous aquifer zone at Site 8 (Figures F4.1-2 and F4.1-3). The coarse-grained aquifer zone material interfingers with fine-grained material in the north-south and east-west cross sections in both the A1- and A2-aquifer zones. Thickness of the aquifer zone material ranges from 2 to 20 feet. The estimated average thickness of the aquifer zone is 15 feet.

#### F.4.1.3 Site-Specific Hydrogeology

Groundwater elevations were calculated from static water level measurements obtained before the constant discharge pump test. Figure F4.1-4 is a potentiometric surface map of the A1-aquifer zone. Local groundwater in the A1-aquifer zone flows to the northeast at an approximate horizontal gradient of 0.004.

#### F.4.1.4 Overview of Testing

A step-drawdown test was conducted on well W08-06(A1-A2) on October 8, 1991 for 4 hours, 6 minutes. Section F.4.1.5 provides a complete discussion of the step-drawdown test.

The constant rate aquifer test was started on November 25, 1991. The pumping well was pumped at 15 gallons per minute (gpm) for approximately 24 hours, followed by a 13-hour recovery period. Thirteen wells, including the pumping well, were monitored during the pump test. The seven wells closest to the pumping well were monitored using an electronic data logger. The remaining wells were monitored manually. Wells monitored during this test are listed in Table F4.1-1. The depth to water observed during the pumping period is given in Attachment I. Figure F4.1-1 shows the location of the monitoring wells and the areas affected by pumping.

#### F.4.1.5 Step-Drawdown Test

The step-drawdown test was conducted over a 4-hour, 6-minute period on October 8, 1991 at well W08-06(A1) to establish a suitable pumping rate for the 24-hour constant discharge pump test. The step-drawdown test at well W08-06(A1-A2) consisted of four steps of pumping at successively higher discharge rates of 2.1, 4.5, 8.5, and 16.5 gpm. The steps ranged from 24 to 120 minutes with a total test time of 366 minutes. Each step was pumped until drawdown stabilized. The relationship between the discharge rate and drawdown for each step is provided in Figure F4.1-5. The drawdown resulting at the end of this test ranged from 0.58 to 4.13 feet with a total drawdown of 6.84 feet. Figure F4.1-5 provides the drawdown-discharge relationships and indicates that if an additional step had been attempted at 33 gpm, the capacity of the well would have been exceeded. Review of the step-draw-

down data revealed that sufficient drawdown could be attained for the constant discharge pump test using a pumping rate of approximately 15 gpm.

#### F.4.1.6 Aquifer Analysis

Values of transmissivity and storativity were calculated from five wells monitored during Pump Test 7. The pumping well was analyzed using the Theis recovery method (Figure F4.1-6), and the observation wells or piezometers were analyzed using the Cooper-Jacob approximation method and the Hantush-Jacob method for semiconfined conditions. The graphical results of the analyses are given in Figures F4.1-6 through F4.1-12.

As judged from Figures F4.1-1 and F4.1-2, the water levels in the A1- and A2-aquifer zones are above the unit tops implying semiconfined or confined conditions. Several of the curves appear to closely approximate curves for Theis solutions; however, a distinct and sharp flattening is apparent in the drawdown versus log time graphs for PZ8.7-1(A1) (Figure F4.1-8), and there is no return to a Theis curve at a later time (greater than 700 minutes). Because of this, the Hantush-Jacob method for semiconfined conditions was chosen for analyzing the aquifer test data. For comparison, the test data for two monitoring wells were also analyzed using the Cooper-Jacob approximation method. Recovery data for the test well are provided in Figure F4.1-6. The datum that is greater than 16 minute passes through the origin, which suggests that there is little change in storage and that recharge effects are minimal.

The transmissivities estimated using the Hantush-Jacob method, assuming a semiconfined aquifer, ranged from 1.00 ft<sup>2</sup>/min (S = 1.1 x 10<sup>-4</sup>) in monitoring well W08-12(A1-A2) to 2.43 ft<sup>2</sup>/min (S = 9.2 x 10<sup>-5</sup>) in piezometer PZ8.7-2(A1). Using an estimated average thickness of 15 feet, these values correspond to a range of hydraulic conductivities of 0.067 to 0.16 ft/min. The complete results of the pump test analysis are presented in Table F4.1-2. Results for both the Hantush-Jacob and the Cooper-Jacob approximation methods for PZ8.7-2(A1) yield similar results. The r/B for this well is relatively small, thus it can be inferred that the leakage is relatively small. In contrast, the results for the two methods applied to the data for PZ8.7-1(A1) do not agree. This may imply greater interconnection between the A1-and A2-aquifer zones in the vicinity of PZ8.7-1(A1).

The intervening A1/A2 aquitard is relatively thin in certain portions of the test site (Figures F4.1-2 and F4.1-3), thus providing evidence of an interconnection between the A-1 and A-2

aquifer zones (Table F4.1-3). The range in transmissivity values from this aquifer test may result from the variable thickness of the aquifer system at this location.

Monitoring well W08-08(A1) was used to monitor site-wide water level changes during Pump Test 7. This monitoring well is 610 feet downgradient of the pumping well. Less than 0.1 foot of fluctuation of water levels was observed in the monitoring well during the aquifer test period. This fluctuation was within the range observed during nonpumping times; however, the major portion of the water level fluctuation during the test paralleled water level changes induced by pumping. These observations suggest a possible pumping influence at the monitoring well along with minor long-term effects facility-wide.

A distance drawdown plot is provided in Figure F4.1-3 along with a best fit line. Using the method presented in Driscoll (1986), transmissivity of the A-aquifer zone is 0.68 ft<sup>2</sup>/min. This value is lower than the values obtained by nonequilibrium methods and may indicate that steady state was not approached. At the farthest monitoring wells clearly affected by the pump test, monitoring wells W08-11(A2) and W08-04(A1), maximum drawdown was 1.8 and 0.17, respectively (Table F4.1-1 and Figure F4.1-13) at the end of the pump test. Some influence from the pump test may be seen at the control well W08-08(A1). At the end of the pumping period, drawdown at the background well W08-08(A1) was 0.7 foot, but decreasing water levels continued at this well until at least 56 minutes after the pump test had been terminated. Background monitoring well W08-08(A1) is downgradient from the pumping well, and a continued decrease in water levels could be expected as the drawdown begins to recover in the vicinity of the production well. Therefore, radial influence in the downgradient direction may be much greater than 300 feet.

## F.4.1.7 Groundwater Quality

Three rounds of sampling were conducted from the pumping well during the pump test. Results indicate that no significant change in contaminant levels occurred during pumping. Aquifer test sampling results for groundwater include TCE concentrations varying from 2 to 7 ppb. 1,1,1-TCA concentrations ranged from 21 to 27 ppb. Dichloroethane (DCA) and dichloroethene (DCE) concentrations averaged approximately 10 ppb (Table F4.1-4).

## F.4.2 Pump Test 1, Site 9

Pump Test 1 was used to assess the A1- and A2-aquifer zones. The objective of Pump Test 1 was to establish whether contaminants may migrate vertically from the A1-aquifer zone to the

A2-aquifer zone in this area. The location of Pump Test 1 was chosen to coincide with the highest TCE concentrations observed at Site 9. A1-aquifer zone monitoring wells had TCE concentrations between 5,000 and 10,000 ppb. The A2-aquifer zone wells within the test area had TCE concentrations between 2,200 and 2,600 ppb (IT, 1991b). An A1-aquifer zone pump test was conducted by a separate contractor at this test site before performance of this pump test (PRC, 1991).

#### F.4.2.1 New Wells/Piezometers Installed

Four piezometers were installed for Pump Test 1 (Table F4.2-1). The piezometers were placed in the A1- and A2-aquifer zones to provide drawdown data in the vicinity of the pumping well (W09-09[A2]) (Figure F4.2-1).

Geologic cross sections showing the relationships of screened zones to hydrogeologic units are shown in Figures F4.2-2 and F4.2-3. Three of the newly installed piezometers were placed in a cluster approximately 10 feet from the pumping well. Piezometers PZ9.1-4(A1) and PZ9.1-2(A2) were installed in the A1- and A2-aquifer zones, respectively. Piezometer PZ9.1-1(AQ) was installed in the A1/A2 aquitard. Piezometer PZ9.1-3(A2) was installed in the A2-aquifer zone approximately 20 feet from the pumping well.

## F.4.2.2 Site-Specific Geology

Site 9, in the vicinity of Pump Test 1, is underlain by aquifer zones A1- and A2-separated by the A1/A2 aquitard (Figures F4.2-2 and F4.2-3). The A1- and A2-aquifer zones occur from approximately 15 to 50 feet below land surface (bls) in the vicinity of Pump Test 1.

The geology within the A1-aquifer zone is characterized as predominantly fine grained with thin, discontinuous transmissive deposits of silty and clayey sand ranging in thickness from 1.5 to 5 feet. The A1 transmissive units are discontinuous and range in depth from approximately 15 to 27.5 feet bls (Figures F4.2-2 and F4.2-3).

The A2-aquifer zone consists of cleaner sands and gravels of relatively greater thickness within a larger clayey and silty body. The sands and gravels range in depth from 28.5 to 47.7 feet bls (Figures F4.2-2 and F4.2-3). The A2 transmissive unit is laterally continuous across the test area in the north-south cross section (Figure F4.2-2). The lateral extent of the A2 unit appears limited to approximately 425 feet in the east-west cross section (Figure F4.2-3). Sand units in the A2-aquifer zone are encountered with highly variable thickness ranging

from approximately 1.5 to 15 feet. The estimated average thickness of the A2-aquifer zone is approximately 12 feet.

The A1/A2 aquitard is observed throughout the Pump Test 1 area. Adjacent to the pumping well, the A1/A2 aquitard lies approximately 25 to 31 feet bls. The aquitard is composed of sandy silt and clay.

## F.4.2.3 Site-Specific Hydrogeology

The A1-aquifer zone can be characterized as confined or semiconfined throughout the area covered by this pump test. Cross sections shown in Figures F4.2-2 and F4.2-3 indicate that static water levels are above the tops of the aquifer zones throughout the pump test at this location. However, the storage coefficients obtained from the aquifer analysis are high for a confined aquifer (F.4.2-2) and suggest that the aquifer zones are semiconfined.

Groundwater elevations were calculated from static water level measurements obtained prior to the constant discharge pump test. Figures F4.2-4 and F4.2-5 are potentiometric surface maps of the A1- and A2-aquifer zones, respectively. Groundwater elevations and gradients in both zones are similar. Local groundwater flow in the A1-aquifer zone is northerly in the vicinity of the pumping well. The approximate horizontal gradient is 0.006. Groundwater flow in the A2-aquifer zone is also northerly in the vicinity of the pumping well with an approximate horizontal gradient of 0.007.

Two well pairs were used to calculate the hydraulic head differences between the A1- and A2-aquifer zones. The results showed an upward flow potential from the A2 to the A1- aquifer zones, with head differences of 0.89 and 0.19 foot. The larger of the two differences was calculated at the pumping well (Table F4.2-3). Note that the potential for flow between the A1- and A2-aquifer zones is opposite to that determined for the Site 8 pump test.

## F.4.2.4 Overview of Testing

A step-drawdown test was conducted on well W09-09(A2) on October 4, 1991 over a 4-hour, 11-minute period using a 1-hp submersible pump. A second step-drawdown test was conducted using a more powerful 2-hp pump on November 22, 1991 for a total of 1 hour, 4 minutes. Results of the step-drawdown tests determined an optimum pumping rate of 42 gpm for the constant discharge pump test. Section F.4.2.5 provides a complete discussion on the

step-drawdown tests. A slug test was performed on the aquitard piezometer PZ9.1-1(AQ) to determine the approximate hydraulic conductivity and storativity of the A1/A2 aquitard.

The pump test was conducted between November 22 and 23, 1991. Recovery from the preceding step-drawdown was at 95 percent of the static water level at the start of the pump test. Pumping well W09-09(A2) was pumped at a constant rate of 42 gpm for approximately 24 hours. Recovery was also monitored over a 12-hour period. The pumping well and 7 monitoring wells and piezometers were monitored for water levels with digital data loggers, and another 13 monitoring wells were monitored with water level probes. Of the wells and piezometers monitored, four were used to provide quantitative estimates of the A2-aquifer zone aquifer parameters. The remaining monitoring wells and piezometers screened in the A1-aquifer zone and in the A1/A2 aquitard were used to qualitatively assess the extent of interconnection between the A1- and A2-aquifer zones. The piezometers and wells monitored during Pump Test 1 are described in Table F4.2-1. Figure F4.2-1 shows the vicinity of the pump test.

## F.4.2.5 Step-Drawdown Test

An initial step-drawdown test was conducted October 4, 1991 at well W09-09(A2). On November 22, 1991, a second step-drawdown test was conducted using a more powerful 2-hp submersible pump. The step-drawdown tests were conducted to establish the optimum production rate for the 24-hour constant discharge test. The step-drawdown data are provided in Figure F4.2-6. Specific capacity for the pumping well is also shown in Figure F4.2-6.

The initial step-drawdown test consisted of six steps pumped at successively higher discharge rates of 1.8, 4.0, 8.0, 14.4, 21.0, and 28.8 gpm. The steps ranged from 30 to 60 minutes for a total test time of 251 minutes and resulted in a total drawdown of 5.12 feet. Dynamic head loss due to friction in the discharge pipe prevented increasing discharge beyond 28.8 gpm using the 1-hp pump. This test was terminated, and a more powerful pump was used.

After installing a higher capacity pump, the step-drawdown test was resumed using discharge rates of 36, 41, 50, and 58 gpm. The steps ranged from 8.5 to 28 minutes with a total test time of 64 minutes. Each step was pumped until drawdown stabilized. The total drawdown at the end of the test was 9.45 feet. Inspection of the plot of drawdown versus time for the step-drawdown test (Figure F4.2-6) suggests that the capacity of the aquifer at this location was not exceeded even at the highest discharge rate. A discharge rate of 42 gpm with a

drawdown of between 7.0 and 8.5 feet was determined to be adequate to sufficiently stress the aquifer at this location.

## F.4.2.6 Aquifer Analysis

Values of transmissivity and storativity were calculated from five monitoring wells and piezometers observed during Pump Test 1. The pumping well was analyzed using the Theis recovery method (Figure F4.2-7), and the Neuman Method for partially penetrating wells in unconfined aquifers (Figure F4.2-7A). Monitoring wells and piezometers were analyzed using the Theis curve method and the Hantush-Jacob method for semiconfined aquifers; the Hantush-Jacob method assumes no storage within the aquitard (Figures F4.2-8 through F4.2-15).

Pumping well W09-09(A2) can be considered to only partially penetrate the A2-aquifer zone. The screened interval begins in the middle of the gravel layer of the aquifer and ends at the silty clay aquitard. To evaluate the effects of partial penetration on the pump test, the Neuman Method was used with partial penetration parameters factored in. Results of the Neuman data were compared to the Theis Well Recovery data for the pumping well.

Transmissivity by the Theis Recovery Method was approximately 1.1 ft²/min while by the Neuman Method it was 0.7 ft²/min. These results were well within an order of magnitude of each other and verify the test data. Given the similar transmissivities and that the effect of partial penetration is negligible on flow pattern and drawdown beyond a radial distance larger than 1.5 to 2 times the saturated thickness (Todd, 1980), the effects of partial penetration on the observation wells are minimal.

A2-aquifer zone transmissivities estimated from monitoring well and piezometer data analyzed using the Hantush-Jacob method ranged from  $0.75 \text{ ft}^2/\text{min}$  (S =  $1.2 \times 10^4$ ) for piezometer PZ9.1-2(A2) to  $4.82 \text{ ft}^2/\text{min}$  (S =  $3.7 \times 10^4$ ) for monitoring well W09-25(A2). Using an estimated average thickness of 12 feet, hydraulic conductivities ranged from 0.063 to 0.40 ft/min. The results of the Pump Test 1 analysis are summarized in Table F4.2-2. The Theis method results were considered to be not as representative of aquifer characteristics and were not used in the summary. Theis data are presented in Table F4.2-2 for comparative purposes.

Comparison of observed response data to the Theis curve solution (confined aquifers) and to leaky type curves (Figures F4.2-8 through F4.2-15) suggests that the A2-aquifer zone receives

recharge or leakage from a boundary layer. Observed drawdowns flatten with time for PZ9.1-2(A2) and PZ9.1-3(A2) suggesting either recharge or delayed yield (Freeze and Cherry, 1979). Because the static water level for the A2-aquifer is above the unit top, delayed yield is not considered likely. Recovery response is significantly displaced to the right (Figure F4.2-7), also indicating recharge. Both Theis- and Hantush-type curves fit response curves for some wells at this location, which suggests that some portions of the A-aquifer system are confined or that only the initial part of the response curve was obtained.

The interconnection of the two aquifers appears significant because pumping of the A2-aquifer zone caused drawdown in A1-aquifer zone monitoring wells that ranged from 1.92 feet in a piezometer 25 feet from the pumping well to 0.77 foot in a monitoring well 220 feet from the pumping well (Figure F4.2-1; Attachment II). Nonzero values of r/B provided by type curve matching with the Hantush semiconfined curves provide further evidence of leakage through the A1/A2 aquitard. Because the time-drawdown plots were closely matched to the type curves that neglect storage in the aquitard, any leakage, if found, can be reasonably assumed to have passed through the aquitard from the A1-aquifer zone. The intervening A1/A2 aquitard is relatively thin in certain portions of the test site (Figures F4.2-2 and F4.2-3), thus providing further evidence of an interconnection between the A1- and A2-aquifer zones.

Monitoring well W09-31(A1) was monitored during the pumping and recovery phases of the test to determine the effects of long-term, site-wide water level changes. During the recovery phase of the test, monitoring wells W09-28(A2) and W09-20(A2) were monitored for the same purpose (Figure F4.2-1). Results of monitoring in monitoring well W09-31(A1) indicated a water level change of 0.02 foot during pumping with no changes during the recovery period. Monitoring of the A2 background monitoring wells revealed a rise in water level of 0.02 foot during the recovery phase of the test. The water level changes in background monitoring wells were approximately 1 percent of induced water level changes and did not have a significant effect on the pump test analysis in piezometers PZ9.1-2(A2) and PZ9.1-3(A2) and monitoring well W09-09(A2).

The pump test influenced wells in the A1-aquifer zone to a maximum distance of 437 feet by the end of the 24-hour test (W09-31[A1]) in Figure F4.2-1). One important aspect of pumping influence at Moffett Field is that some more distant wells responded with more drawdown than those wells closer to the pump (e.g., W09-25[A2] and W09-34[A2]. This

may be the result of aquifer heterogeneity (Figures F4.2-2 and F4.2-3) under the influence of existing hydraulic gradients (Figures F4.2-4 and F4.2-5). Figure F4.2-16 is a semilog plot of distance and drawdown, which can be used to provide an estimate of the overall transmissivity of the aquifer zones. Transmissivity values computed by this method yielded a result of 0.50 ft<sup>2</sup>/min. This value is slightly lower than the lowest values given by single well analysis.

## F.4.2.7 Slug Test Analysis

A slug test was performed on the aquitard piezometer PZ9.1-1(AQ) in conjunction with two other aquitard piezometer slug tests at Site 9 (Sections F.4.3.7 and F.4.5.7). The calculated transmissivity and storativity can only be used for qualitative characterization of the aquitard because of site conditions (Cooper, et al., 1967). Of the three slug tests performed on the aquitard at Site 9, this well produced the lowest calculated values of hydraulic conductivity and was approximately the same as the vertical hydraulic conductivity computed from the consolidation data provided below.

Using an assumed aquitard thickness of 5.5 feet, the estimated horizontal hydraulic conductivity is 9.1 x  $10^{-5}$  ft/min (Figure F4.2-17). The estimated value of transmissivity is 5.0 x  $10^{-4}$  ft<sup>2</sup>/min, and the estimated storativity is 1 x  $10^{-5}$ .

# F.4.2.8 Aquitard Analysis

Piezometer PZ9.1-1(AQ) was placed in the A1/A2 aquitard to measure hydraulic response during pumping, hydraulic response to an instantaneous slug of water (slug test), and geotechnical parameters of the aquitard material.

The consolidation testing of a sample from the PZ9.1-1(AQ) boring was based on ASTM Method 2435. The vertical hydraulic conductivity at the piezometer PZ9.1-1(AQ) was calculated to be  $1.4 \times 10^{-4}$  ft/min. Calculations are presented in Table F4.2-4.

# F.4.2.9 Groundwater Quality

Two rounds of sampling were conducted from the pumping during the pump test. Results indicated a slight insignificant decrease in contaminant concentration in response to pumping (Table F4.2-5). Aquifer test sampling results for groundwater showed TCE concentrations decreasing from 1,900 to 1,800 ppb during the test. Methylene chloride concentrations decreased from 230 to 200 ppb during the test. 1,2-DCE concentrations increased from 56 to

60 ppb; 1,1-DCE stayed at 24 ppb; 1,1-DCA and 1,1,1-TCA concentrations decreased from 25 and 13 ppb to nondetected concentrations (Table F4.2-5).

## F.4.3 Pump Test 3, Site 9

Pump Test 3 assessed the A2-aquifer zone and the A1/A2 aquitard through pumping in the A2-aquifer zone. This pump test site was chosen to characterize the A1- and A2-aquifer zones in an area that is downgradient of a suspected source of fuel contamination and that is also within the regional TCE plume at Site 9 (Figure F4.3-1). TCE was detected in well W09-28(A2) at 17,000 ppb (IT, 1991b). The pumping well is approximately 73 feet downgradient of free-product well FP9-1, which showed soil contamination below the water table from compounds indicative of jet fuel. Similarly, jet fuel-related contaminants were detected in shallow soil samples from pumping well W09-22(A2).

An A1-aquifer zone pump test was originally designed for this location; however, the A1-aquifer zone pump test was not performed, based on the lack of sands in the A1-aquifer zone and the low aquifer hydraulic conductivity values from a previous aquifer test performed on an adjacent A1-aquifer zone monitoring well, W29-02(A1) (PRC, 1991).

## F.4.3.1 New Wells/Piezometers Installed

Five piezometers were installed for Pump Test 3 (Table F4.3-1). The piezometers were placed to provide drawdown data in the vicinity of the pumping well (W09-22[A2]), which fully penetrates the A2-aquifer zone. The A1-aquifer zone piezometer intercepts a 3-foot-thick clayey sand unit in the A1-aquifer zone (Figures F4.3-1 and F4.3-2). Two A2 piezometers, PZ9.3-3(A2) and PZ9.3-5(A2), were placed to fully penetrate the A2-aquifer zone. The aquitard piezometer PZ9.3-1(AQ) is screened over a 1-foot interval within the upper half of the aquitard, which ranges from 11 feet in thickness at the pumping well to 13 feet at the piezometer cluster (Figure F4.3-3). The aquitard piezometer was screened in a silty clay.

#### F.4.3.2 Site-Specific Geology

The A1-aquifer zone is characterized by predominantly fine-grained soils containing interfingering deposits of clean to silty or clayey sand. Interfingering sands in the A1-aquifer zone appear discontinuous in the north-south cross section south of pumping well W29-09(A2) (Figure F4.3-2). Sands of the A1-aquifer zone appear more continuous in the east-west cross section, particularly to the east of pumping well W29-09(A2) (Figure F4.3-3). The thickness

of the A1-aquifer zone in the vicinity of Pump Test 3 varies from 1 to 11 feet. The permeable zones in the A1-aquifer zone occur in a depth range of approximately 15 to 30 feet bls.

The A2-aquifer zone consists of cleaner sands and gravels of relatively greater thickness within a larger fine-grained body. The permeable zone sands and gravels range in depth from 39 to 50 feet bls (Figures F4.3-2 and F4.3-3). The A2 permeable zone appears discontinuous in the north-south cross section (Figure F4.3-2); however, the unit is more continuous in the east-west cross section (Figure F4.3-3). The A2 permeable zones range in thickness from approximately 3 to 10 feet. The estimated average thickness is approximately 6 feet.

The A1/A2 aquitard appears continuous across the test area. This aquitard zone lies approximately 28 to 40 feet bls with thickness depending on the continuity of the A1- and A2-aquifer zones (Figures F4.3-2 and F4.3-3). The aquitard consists of sandy or silty clay.

## F.4.3.3 Site-Specific Hydrogeology

In the vicinity of Pump Test 3, the A1-aquifer zone is confined to semiconfined. The A2-aquifer zone appears to be confined throughout the test area and appears to be more continuous and extensive than the A1-aquifer zone (Table F4.3-2). Aquifer testing suggests that these zones are well connected and appear to exhibit recharge effects.

Groundwater elevations were calculated from static water level measurements obtained prior to the constant discharge pump test. Figures F4.3-4 and F4.3-5 are potentiometric surface maps of the A1- and A2-aquifer zones, respectively. Groundwater gradients in both zones are similar. Local groundwater flow in the A1-aquifer zone is northeasterly in the vicinity of the pumping well. The approximate horizontal gradient is 0.007. Groundwater flow in the A2-aquifer zone is also northerly with an approximate horizontal gradient of 0.005.

Pump Test 3 did not have A1/A2-aquifer zone well pairs. To calculate the vertical difference, monitoring wells W09-23(A1) and W09-28(A2) that were located 95 feet apart were chosen. The result was an upward gradient from the A2- to the A1-aquifer zone with a head difference of 0.30 foot (Table F4.3-3).

## F.4.3.4 Overview of Testing

A step-drawdown test was conducted on well W09-22(A2) on October 23, 1991 for a total of 2 hours and 48 minutes. Section F.4.3.5 provides details on the step-drawdown test.

A slug test was performed on the aquitard piezometer PZ9.3-1(AQ) to estimate the hydraulic conductivity of the A1/A2 aquitard. This test was conducted with two other aquitard slug tests (Sections F.4.2.7 and F.4.5.7).

On November 7, 1991, the aquifer test was begun on monitoring well W09-22(A2) with a constant discharge rate of 15 gpm. The test began at 14:35 hours after two false starts. The well was pumped for approximately 24 hours, and recovery was monitored for approximately 10 hours. The pumping well and 7 monitoring wells were monitored with a data logger, and another 13 wells were monitored for water levels with calibrated water level probes. Table F4.3-1 summarizes the geometries of wells and piezometers observed during Pump Test 3. Figure F4.3-1 shows well locations. Quantitative analyses were performed on the pumped well W09-22(A2), on deepest piezometers PZ9.3-3(A2) and PZ9.3-5(A2), and on monitoring well W29-08(A2). Methods and results of analysis are summarized in Table F4.3-2. The remaining monitoring wells and piezometers were monitored to provide qualitative information on interconnection within the A1- and A2-aquifer zones.

#### F.4.3.5 Step-Drawdown Test

The step-drawdown test was conducted on October 23, 1991 at monitoring well W09-22(A2) to determine a production rate for the 24-hour constant discharge test. Analysis of the step-drawdown test data provided values of specific capacity that were then evaluated to determine an optimal pumping rate. The step-drawdown test at well W09-22(A2) consisted of three steps of pumping at successively higher discharge rates of 5, 10, and 16.2 gpm. The steps ranged from 34 to 90 minutes with a total test time of 168 minutes. With the exception of the third step, each step was pumped until drawdown stabilized. A table of the discharge rate and drawdown for each step is given in Figure F4.3-6. The drawdown resulting from the three steps of this test ranged from 1.45 to greater than 10.2 feet. Total drawdown for the step-drawdown test was greater than 13.8 feet. Drawdown data for the third step cannot be used quantitatively but suggests that the pumping well capacity was very close to exceeding a discharge rate of 16.2 gpm. A discharge rate of 15 gpm was determined to be optimal for the constant rate pump test.

## F.4.3.6 Aquifer Analysis

Values of transmissivity and storativity were calculated from four wells and piezometers at Pump Test Site 3. Analytical methods and results are summarized in Table F4.3-2. The pumping well was analyzed using the Theis recovery method (Figure F4.3-7). The best fit line for the recovery does not intersect the origin but is displaced significantly to the right, suggesting a significant recharge effect.

Data from each monitoring well and piezometer included in the quantitative analysis were analyzed using the Theis method and the Hantush-Jacob method for semiconfined aquifers. In applying the Hantush-Jacob method, aquitard storage was assumed to be zero. This assumption was based on the observations that drawdowns approached equilibrium relatively rapidly and that delayed yield response was not observed.

Comparison of observed data to the Theis curve for confined aquifers (Figures F4.3-8, F4.3-10, and F4.3-12) and to Hantush-Jacob type curves for semiconfined aquifers (Figures F4.3-9, F4.3-11, and F4.3-13) strongly suggests that the A2-aquifer zone near pumping well W09-22(A2) is semiconfined. Observed drawdowns approach steady state with time, implying recharge from a bounding layer. Lateral recharge boundaries capable of supporting steady-state drawdowns within the duration of the pump test are also considered unlikely.

A2-aquifer zone transmissivities estimated using the method of Hantush-Jacob ranged from 0.29 ft<sup>2</sup>/min (S = 6.8 x 10<sup>-5</sup>) in piezometer PZ9.3-5(A2) to 0.53 ft<sup>2</sup>/min (S = 1.5 x 10<sup>-3</sup>) in monitoring well W29-08(A2). Using an estimated average thickness of 6 feet (Section F.4.3.3, Figures F4.3-2 and F4.3-3), hydraulic conductivities ranged from 0.047 to 0.088 ft/min. The complete results of the pump test analysis are summarized in Table F4.3-2. As discussed previously, stated results of the Theis curve analysis are not considered representative of aquifer conditions and are presented in Table F4.3-2 for comparative purposes.

One background well, W09-35(A1), located approximately 650 feet southeast of the pumping well, was monitored with a water level probe for possible site-wide water level changes that may have affected test results. The control well, screened within the A1-aquifer zone, exhibited a drop of 0.04 foot during pumping and a rise of 0.03 foot during recovery. The changes in water level observed in the control well were approximately 3 to 4 percent of drawdown induced by pumping in monitoring well W29-08(A2). Monitoring well W29-08(A2) is located 111 feet from the pumped well and was the most distant observation point

used for quantitative analysis of aquifer parameters. Based on these observations, the change in drawdown attributable to variation in site-wide water levels is not considered significant to the pump test analyses. A semilog plot of distance and drawdown is provided in Figure F4.3-14. Transmissivity of the aquifer can be computed from the drawdown across one log cycle and the discharge rate (Driscoll 1986). This method yields a transmissivity of 0.12 ft<sup>2</sup>/min. This value is slightly lower than the values for T computed using Theis and Hantush-Jacob methods.

The pump test provided a drawdown of 0.1 foot in more distant wells at a maximum distance of 383 feet from the pumping well by the end of the 24-hour test. Some wells responded with more drawdown than those wells closer to the pumping well (Figure F4.3-14). Also, the resulting shape of the area of influence is irregular (Figure F4.3-1). This is most likely the result of permeable zones (sand channels) within the aquifer material and/or complex stacking of several permeable (sand) deposits.

## F.4.3.7 Slug Test Analysis

A slug test was performed on the aquitard piezometer for PZ9.3-1(AQ). The test was performed in conjunction with two other aquitard piezometer slug tests (Sections F.4.2.7 and F.4.5.7). The reported transmissivity and storativity should be used with caution because the assumptions inherent in the analysis may not reflect field conditions (Cooper, et al., 1967).

Based on the slug test analysis, the estimated value of transmissivity was  $3.8 \times 10^{-3} \text{ ft}^2/\text{min}$  and was calculated with a storativity value of  $5 \times 10^{-5}$ . Using an assumed thickness of 17 feet, the estimated value of hydraulic conductivity is  $2.3 \times 10^{-4}$  ft/min (Figure F4.3-15).

## F.4.3.8 Aquitard Analysis

Piezometer PZ9.3-1(AQ) was placed in the A1/A2 aquitard to measure hydraulic response during pumping, hydraulic response to an instantaneous slug of water, and geotechnical parameters of the aquitard soil.

The consolidation testing of a soil sample from the PZ9.3-1(AQ) boring was based on ASTM Method 2435.

The calculated value of the aquitard specific storage (S<sub>s</sub>) from the sample was 1.95 x 10<sup>-4</sup> ft<sup>-1</sup>. This value was used in Neuman-Witherspoon's ratio method of aquitard analysis to arrive

at a value of vertical hydraulic conductivity. Values of drawdown and calculations are presented in Table F4.3-4. The calculated vertical hydraulic conductivity (K') for the piezometer location is  $1.84 \times 10^{-4}$  ft/min.

## F.4.3.9 Groundwater Quality

Three rounds of sampling were conducted during this pump test. Results indicate an insignificant increase in the concentrations of some VOCs. Aquifer test sampling results for groundwater TCE concentrations ranged from 3,200 to 3,900 ppb during the test. Methylene chloride concentrations ranged from 110 to 210 ppb during the test (Table F4.3-5).

## F.4.4 Pump Test 5, Site 9

Pump Test 5 assessed the A1- and A2-aquifer zones with independent A1- and A2-aquifer zone pump tests. The location of the pump tests was chosen to characterize the A1- and A2-aquifer zones in an area affected by the regional VOC plume. During quarterly sampling at monitoring well W09-38(A1), TCE was detected at 5,800 ppb; at A2-aquifer monitoring well W09-41(A2), TCE was detected at 12,000 ppb (IT, 1991b). The pump tests at this location were also intended to provide data for determining the degree of interconnection between the A1- and A2-aquifer zones in this portion of Site 9 and to identify possible preferred migration paths from upgradient or cross gradient sources, including Building 88 (Figure F4.4-1).

#### F.4.4.1 New Wells/Piezometers Installed

Seven piezometers were installed to meet the objectives of Pump Test 5 (Table F4.4-1). Two new pumping wells (PT9-2[A1] and PT9-3[A2]) were installed as a pair but were used as piezometers due to low yields in the A2-aquifer zone during development. Monitoring wells W09-38(A1) and W09-41(A2) were chosen as pumping wells (Figures F4.4-1 and F4.4-2).

Three A1-aquifer zone piezometers (PZ9.5-4[A1], PZ9.5-6[A1], and PT9-2[A1]) were installed to monitor the A1-aquifer zone (Figures F4.4-1, F4.4-3, and F4.4-4). Piezometers were placed to provide drawdown data in the vicinity of the pumping wells.

Three A2-aquifer zone piezometers were installed. Piezometer PZ9.5-5(A2) was placed to fully penetrate a permeable zone (sand channel) below the pumped zone. Piezometers PZ9.5-7(A2) and PT9-3(A2) were placed to fully penetrate the A2-aquifer zone (Figures F4.4-3 and F4.4-4).

The intended aquitard piezometer PZ9.5-1(AQ) was placed in a cluster with piezometers PZ9.5-4(A1) and PZ9.5-5(A2) southeast of the pumping wells (Figure F4.4-1). The aquitard piezometer was screened over a 1-foot interval and intercepted a transmissive sand unit adjacent to the A2-aquifer zone pumping well screen interval (Figure F4.4-4). For this reason, it has been treated as an A2-aquifer zone piezometer.

## F.4.4.2 Site-Specific Geology

The A1-aquifer zone is characterized by relatively thick transmissive silty to clean sand and gravel deposits within a silty clay body. The A1-aquifer zone is continuous across the pump test site area in the north, south, and western direction. Laterally in the eastern direction, the A1-aquifer zone appears to be continuous based on limited subsurface data (Figure F4.4-4). The A1 permeable zone (sand channels) ranges in thickness from approximately 10 to 21.5 feet (Figures F4.4-3 and F4.4-4). The A1 permeable zones occur at depths ranging from approximately 9 to 30 feet bls. The estimated average thickness is 13 feet.

The A2-aquifer zone consists of thin sand and gravel deposits with relatively high amounts of silt within a silty clay body. The A2-aquifer zone sands and gravels occur at depths between 35 and 42 feet bls in well W09-41(A2) and between 30.5 and 40 feet bls in well W09-08(A2). The A2 unit ranges in thickness from 4 to 10 feet with an estimated average thickness of 6 feet. The A2-aquifer zone is laterally discontinuous over the test site area (Figures F4.4-3 and F4.4-4).

The A1/A2 aquitard appears continuous across the entire area. The aquitard lies approximately 30 to 33 feet bls and is composed of clayey silt with sand, and silty clay with sand (Figures F4.4-3 and F4.4-4).

## F.4.4.3 Site-Specific Hydrogeology

The A1-aquifer zone in the vicinity of Pump Test 5 (Site 9) appears to be semiconfined (Figures F4.4-3 and F4.4-4); however, the boring log of pumping well W09-38(A1) reveals the presence of clayey sands at approximately the same elevation as the potentiometric surface (Table F4.4-2). In the boring log, these clayey sands are separated from the primary A1-aquifer zone by a thin clay. Although the water levels in all other A1-aquifer zone monitoring wells and piezometers shown in Figures F4.4-3 and F4.4-4 indicate semiconfined conditions, the stratigraphy in the vicinity of the pumping wells demonstrates that locally unconfined conditions are possible.

As illustrated in Figures F4.4-3 and F4.4-4, water levels in piezometers and wells screened in the A2-aquifer zone are at a higher elevation than the A2-aquifer zone. Based on this observation and on the presence of numerous sandy layers, the A1- and A2-aquifer zones are semiconfined.

Groundwater elevations were calculated from static water level measurements obtained prior to the constant discharge pump test. Figures F4.4-5 and F4.4-6 are potentiometric surface maps of the A1- and A2-aquifer zones, respectively. Local groundwater flow in the A1-aquifer zone is northeasterly in the vicinity of the pumping well. The approximate horizontal gradient is 0.005. Groundwater flow in the A2-aquifer zone is northerly with an approximate horizontal gradient of 0.006.

One well pair (W09-38[A1] and W09-41[A2]) was used to calculate the vertical hydraulic head differences between the A1- and A2-aquifer zone levels. The flow potential is upward from the A2 to the A1-aquifer zone with a head difference of 0.20 foot (Table F4.4-3). This flow potential is consistent with other well pairs at Site 9.

## F.4.4.4 Overview of Testing

## F.4.4.4.1 A1-Aquifer Zone

An initial step-drawdown test was conducted on the A1-aquifer zone pumping well W09-38(A1) on October 25, 1991 for 3 hours, 26 minutes using a 1-hp pump. A second step-drawdown test using a more powerful 2-hp pump was conducted on the same well on December 4, 1991 for 1 hour, 20 minutes. Results of the second step-drawdown test indicated that the optimum pumping rate for the constant discharge pump test in the A1-aquifer zone was 40 gpm. See Section 4.4.5.1 for complete details of the step-drawdown test.

A planned slug test was not performed on piezometer PZ9.5-1(AQ) because the screen interval was placed in a small sand unit rather than the A1/A2 aquitard. This piezometer was treated as an A2 piezometer.

The A1-aquifer zone pump test began on December 4, 1991. Water levels affected by the step-drawdown test were allowed to return to static levels before the pump test began. Pumping well W09-38(A1) was pumped at 40 gpm for approximately 27 hours. The test site was also monitored for a 14-hour, 30-minute recovery period. The pumping well and 6

monitoring wells were monitored with transducers, and another 14 monitoring wells were monitored for water levels with calibrated water level probes. Table F4.4-1 summarizes the geometries of wells and piezometers observed during Pump Test 5. Monitoring well and piezometer locations are shown relative to other Site 9 features in Figure F4.4-1. Quantitative analyses were performed on pumped well W09-38(A1) and on piezometers PZ9.5-4(A1), PZ9.5-6(A1), and PT9-2(A1). Analytical methods and results are summarized in Table F4.4-2. The remaining monitoring wells and piezometers were monitored to provide qualitative information on an interconnection between the A1- and A2-aquifer zones and on possible heterogeneities within the A1- and A2-aquifer zones.

## F.4.4.4.2 A2-Aquifer Zone

A step-drawdown test was conducted on well W09-41(A2) on October 29, 1991 for 5 hours, 40 minutes. This step-drawdown test established an optimal pump rate of 5 gpm for the A2 test. See Section 4.4.5.2 for a complete discussion of the step-drawdown test.

The A2-aquifer zone pump test began on December 2, 1991. Monitoring well W09-41(A2) was pumped at 5 gpm for approximately 25 hours. The pump test site was also monitored for a 13-hour recovery period. The pumping well and 6 monitoring wells were monitored with transducers, and another 12 wells were monitored with water level probes. Monitoring well and piezometer locations are shown relative to the Site 9 features in Figure F4.4-2. Quantitative analyses were performed on pumped well W09-41(A2) and on piezometers PZ9.5-7(A2), PZ9.5-1(AQ), and PT8-3(A2). Analytical methods and results are summarized in Table F4.4-2. The remaining monitoring wells and piezometers were monitored to provide qualitative information on possible A1- and A2-aquifer zone interconnection and on possible heterogeneities within the A1- and A2-aquifer zones.

## F.4.4.5 Step-Drawdown Test

#### F.4.4.5.1 A1-Aquifer Zone

Step-drawdown tests were conducted on October 25, 1991 and December 4, 1991 at well W09-38(A1). The step-drawdown tests were conducted to establish an optimum pumping rate for the 24-hour constant discharge test. The initial step-drawdown test consisted of four steps of pumping at 3.5, 7.2, 15, and 31 gpm with steps ranging from 30 to 90 minutes in duration and resulting in a 3.95-foot drawdown. Due to dynamic head loss in the discharge line and the unexpectedly high water production capacity of the formation, the discharge rates required

to stress the aquifer could not be attained utilizing the 1-hp pump; therefore, the test was terminated and a higher capacity pump was obtained.

The second phase of the step-drawdown test consisted of four more steps at discharge rates of 31, 37, 41.5, and 50 gpm. The steps ranged from 16 to 22 minutes with a total test time of 80 minutes. Each step was terminated when the drawdown time curve began to flatten. A table of the discharge rate and drawdown for each step of the second step test is given in Figure F4.4-7. The total drawdown at the end of the test was 7.85 feet. From the results of the step-drawdown test, a discharge rate of 40 gpm was chosen as adequate for the constant discharge aquifer test.

## F.4.4.5.2 A2-Aquifer Zone

A step-drawdown test was conducted on October 29, 1991 at well W09-41(A2) to determine the optimum pumping rate for the 24-hour constant discharge test. A step-drawdown test at well W09-41(A2) consisted of four steps, three of which were pumped at successively higher discharge rates of 2, 4, and 8 gpm. The fourth step was pumped at 5.4 gpm because the third discharge rate (8 gpm) caused encroachment on the well screen. The steps ranged from 30 minutes to 2 hours with a total test time of 5 hours and 40 minutes. Drawdown stabilized during Steps 1 and 2. A table of the discharge rate and drawdown for each step is provided in Figure F4.4-8. The drawdown resulting from the three steps of this test ranged from 2 feet to greater than 21 feet with a total drawdown of more than 23 feet. Drawdown data for the third step cannot be used quantitatively because stabilization for this step was not achieved. Review of the data, although limited, indicates that a 5 gpm discharge rate would adequately stress the aquifer during the constant discharge pump test.

# F.4.4.6 Aquifer Analysis

# F.4.4.6.1 A1-Aquifer Zone

Transmissivity and storativity were estimated from data obtained at two piezometers and one monitoring well using the Theis method (Table F4.4-2). Transmissivity was estimated for the pumping well using the Theis recovery method (Figure F4.4-9). The best fit line defined by the recovery data does not intercept the origin but is displaced to the left, suggesting incomplete recovery, perhaps due to a limited extent of the aquifer.

The range of estimated transmissivities in the A1-aquifer zone monitoring well and piezometers varied between 2.58 ft<sup>2</sup>/min ( $S = 1.7 \times 10^{-3}$ ) in monitoring well PT9-2(A1) and 4.16 ft<sup>2</sup>/min ( $S = 3.9 \times 10^{-3}$ ) in piezometer PZ9.5-4(A1). Assuming an estimated average aquifer zone thickness of 13 feet, the hydraulic conductivities ranged from 0.20 ft/min to 0.32 ft/min. Transmissivity at the pumping well (W09-38[A1]) was estimated to be 2.07 ft<sup>2</sup>/min (Table F4.4-2). The transmissivities estimated for the observation points are considered to be overestimates due to a marked deviation of the observed data from the model-based Theis curve. Justifications for the use of the Theis method are provided in the following section.

Figures F4.4-10, F4.4-11, and F4.4-12 present time-drawdown data for the A1-aquifer zone monitoring well and piezometers plotted with the Theis master curve. Each of the three sets of observed data shows a marked positive deviation of the curve relative to the Theis curve at approximately 100 minutes. Application of the Neuman (1975) method for unconfined aquifers resulted in a poor match between observed data and theoretical curves, chiefly because a delayed yield response was not apparent in the observed data. The Hantush-Jacob method for semiconfined (leaky) aquifers was ruled out because the observed data deviated and did not approach steady state as assumed by the model. Deviation upwards from the Theis curve suggests reduction in T and S laterally away from the pumping well. These considerations lead to the presentation of the observed data with the Theis master curve.

The observed deflection of the observed data from the Theis curve is consistent with lateral changes in T and S within the aquifer. As shown in Figures F4.4-3 and F4.4-4, the maximum thickness of the A1-aquifer zone in the vicinity of Pump Test 5 is approximately 22 feet. The maximum thickness of the aquifer zone appears to occur at the pumping well. Away from the pumping well, observed thicknesses are reduced to approximately 10 feet. In the east-west cross section, the zone may not be continuous (Figure F4.4-4) and, therefore, appears to indicate lateral thinning of the aquifer for the observed data. A thinning of the aquifer is compatible with the displacement of the recovery data and incomplete recovery.

The relatively high estimated transmissivities resulting from the use of the Theis method may be due to the greater thickness of the A1-aquifer zone in the vicinity of the pumped well. The observed deviation of the data from the Theis curve at later times may be due to the thinning of the A1-aquifer zone away from the pumped well. As the zone of depression expanded, it encountered thin or discontinuous portions of the A1-aquifer zone, resulting in drawdown greater than that predicted by the Theis curve.

The range of estimated storativities previously cited is not characteristic of confined or unconfined conditions (Freeze and Cherry, 1979). The one storativity outlier with respect to values expected for confined conditions may be due to model bias, in which case actual A1-aquifer zone storativities may be lower. Another explanation is that the estimated values are representative of actual semiconfined conditions in which aquifer material underwent nonrecoverable aquifer deformation in response to pumping. This could be expected because of the fine-grained nature of a significant fraction of the aquifer material and the consolidated nature of the aquifer material.

Two background monitoring wells were used to monitor regional changes in static water levels during the pumping and recovery phases of the A1-aquifer zone pump test. Background well W09-35(A1), located approximately 800 feet from the pumped well (Figure F4.4-13), exhibited a 0.05-foot decrease in water level during the pumping phase of the test followed by a further decrease of 0.01 foot during the recovery phase of the test. Monitoring well W09-20(A2) was similar in response with a 0.03-foot decrease in water level during pumping and a 0.01-foot decrease during the recovery phase. Background well W09-20(A2) was located approximately 800 feet from the pumping well (Figure F4.4-1). As shown in Figure F4.4-1, the three observation points used to collect data for the quantitative analysis were located within 223 feet of the pumped well and experienced drawdowns of 1 foot or greater. Although a site-wide change in water level appears to have affected the A1- and A2-aquifer zones, the magnitude of the change was less than approximately 5 percent of that due to the pump test and is considered to have had a negligible impact on the results.

The pump test influenced wells at a maximum distance of 507 feet by the end of the 24-hour test. As was the case for most pumping influence at Moffett Field, some site wells responded with more drawdown than those wells closer to the pump (Figure F4.4-13).

Distance drawdown plots can be used to estimate an aquifer's transmissivity from the drawdown over one log cycle of distance and the discharge rate (Driscoll, 1986). Using this method yields a value of 0.82 ft<sup>2</sup>/min, which is lower than transmissivity computed from single well analysis.

## F.4.4.6.2 A2-Aquifer Zone

Transmissivity was estimated at the pumping well using the Theis recovery method (Figure F4.4-14) and the Theis Method with partial penetration (Figure F4.4-14A). Using the Theis

Recovery Method, data from the pumping well fits a line that appears to be displaced to the right suggesting that either storage has decreased or that a recharge effect was present. Transmissivity using the recovery data is 0.29 ft<sup>2</sup>/min. Pumping data were evaluated using the Theis Method with partial penetration parameters factored in to account for the partial screening within the aquifer. Transmissivity using the pumping data with the partial penetration parameters is 0.28 ft<sup>2</sup>/min. Given that the transmissivity values from the two Theis Methods are essentially the same, the test data have been validated.

A2-aquifer zone transmissivity and storativity estimates were determined from data obtained at one monitoring well and two piezometers using the Theis method (Figures F4.4-15, F4.4-17, and F4.4-19) and the Hantush-Jacob method for semiconfined aquifers (Figures F4.4-16, F4.4-18, and F4.4-20). Results are summarized in Table F4.4-2. Based on site stratigraphy and the character of the observed data, the A2-aquifer zone is considered semiconfined. Results of the Hantush-Jacob analysis are considered representative of A2-aquifer zone conditions. Results of the Theis method are provided for comparison.

The range of estimated transmissivities at the A2-aquifer zone monitoring well and deepest piezometers varied between  $0.038 \text{ ft}^2/\text{min}$  (S =  $2.8 \times 10^{-3}$ ) in piezometer PZ9.5-7(A2) and  $0.78 \text{ ft}^2/\text{min}$  (S =  $1.6 \times 10^{-3}$ ) in monitoring well PT9-3(A2). Using an estimated average thickness of 6 feet, the corresponding range of hydraulic conductivities is  $6.3 \times 10^{-3}$  to 0.13 ft/min.

Time versus drawdown plots displayed significant flattening of the drawdown trend. This could be caused by either leakage from adjacent aquitard(s), a recharge boundary, or water released out of storage from newly dewatered material (delayed yield). The values of r/B provided on the curve match plots of the Hantush semiconfined curves (Figures F4.4-16, F4.4-18, and F4.4-20) indicate aquitard leakage. Because the time-drawdown plots were closely matched to the type curves that neglect storage in the A1/A2 aquitard, the leakage can be reasonably assumed to have passed through the aquitard from an overlying (or underlying) aquifer.

Monitoring wells W09-18(A1) and W09-17(A2) were used as control wells in the A1- and A2-aquifer zones, respectively. As shown in Figure F4.4-2, W09-18(A1) is located approximately 600 feet northeast of the pumped well. W09-17(A2) is located approximately 400 feet northeast of the pumped well. The water level in W09-18(A1) decreased by 0.03 foot during

the pumping phase and increased by 0.02 foot during recovery. A similar response, possibly indicative of influence by the pumping well, was observed in monitoring well W09-17(A2) where a water level decline of 0.04 foot during pumping was followed by a 0.01-foot increase during the recovery phase. As illustrated in Figure F4.4-21, which shows water level declines as a function of radial distance from the pumped well, the magnitude of possible site-wide water level changes observed in control wells is negligible.

Pumping influence for the A2-aquifer zone pump test closely mirrored the A1-aquifer zone pump test with smaller values of drawdown and a slightly smaller area of influence (Figure F4.4-2). The pump test influenced wells at a maximum distance of 492 feet by the end of the 24-hour test (Figure F4.4-21). Transmissivity estimated from the distance drawdown plot is 0.11 ft<sup>2</sup>/min, which is comparatively close to values estimated from single well analysis.

# F.4.4.7 Groundwater Quality

## F.4.4.7.1 A1-Aquifer Zone

Three rounds of groundwater sampling were conducted during the A1 pump test. Results did not indicate obvious trends in contaminant concentrations during pumping. Aquifer test sampling results for groundwater TCE concentrations ranged from 3,600 to 4,800 ppb during the test. 1,1-DCE concentrations ranged from 22 to 150 ppb (Table F4.4-4).

#### F.4.4.7.2 A2-Aquifer Zone

Three rounds of sampling were conducted during the A2 pump test. Results did not indicate significant trends of contaminant concentrations. TCE concentrations ranged from 8,000 to 9,900 ppb during the test. 1,1-DCE concentrations ranged from 88 to 110 ppb (Table F4.4-5).

## F.4.5 Pump Test 8, Site 9

Pump Test 8 was conducted to assess the A1- and A2-aquifer zones and the intervening A1/A2 aquitard through separate pump tests in the A1- and A2-aquifer zones. The location of the tests was chosen to characterize the A1- and A2-aquifer zones in the area directly downgradient of a suspected contaminant source at Building 88. This portion of Site 9 is also within the high concentration portion of the regional TCE groundwater plume. Wells W09-35(A1), W09-02(A1), W09-38(A1), and MEW-82(A1) make up a group of wells showing TCE concentrations between 5,000 and 10,000 ppb in the A1-aquifer zone (IT, 1991b). The most downgradient monitoring well of this group (W09-35[A1]) was pumped for the A1-

aquifer zone constant discharge test. Similarly, the A2-aquifer zone regional contaminant plume has extended to this area. The A2-aquifer zone test vicinity is located downgradient of the highest concentration of the A2-aquifer zone plume, which is centered around wells W09-14(A2) and MEW-4(A2) (IT, 1991b). The pumping wells are located approximately 400 feet downgradient of W09-14(A2).

## F.4.5.1 New Wells/Piezometers Installed

Seven piezometers were installed to assess the A1- and A2-aquifer zones and the intervening A1/A2 aquitard (Table F4.5-1 and Figures F4.5-1 and F4.5-2). A1-aquifer zone piezometers PZ9.8-2(A1), PZ9.8-4(A1), and PZ9.8-6(A1) were placed to fully penetrate the A1-aquifer zone. Three A2 piezometers, PZ9.8-3(A2), PZ9.8-5(A2), and PZ9.8-7(A2), were placed to fully penetrate the A2-aquifer zone. The aquitard piezometer PZ9.8-1(AQ) was placed in a cluster with piezometers PZ9.8-2(A1) and PZ9.8-3(A2) and was screened over a 1-foot-thick interval within the A1/A2 aquitard.

#### F.4.5.2 Site-Specific Geology

The A1-aquifer zone is characterized as a predominately sandy to silty clay unit intercalated with transmissive deposits of silty sands and gravels. In the north-south cross section, the A1 transmissive unit thins out to the south, and its northern extent is unknown (Figure F4.5-3). The A1 permeable zone (sand channels) appears laterally continuous beneath the western half of the site in the east-west cross section (Figure F4.5-4). The A1-aquifer zone permeable zone varies significantly in thickness from 2.5 to 21 feet. The estimated average thickness is 8 feet.

The A2-aquifer zone consists of thin interfingering silty sands and gravels occurring at depths ranging from 28 to 45.5 feet bls (Figures F4.5-3 and F4.5-4). The A2-aquifer transmissive zone appears laterally continuous in the east-west cross section but may not be continuous in the north-south cross section. The range of thickness of the A2-aquifer transmissive unit is 4 to 18.5 feet (Figure F4.5-4). The estimated average thickness is 10 feet.

The A1/A2 aquitard appears continuous in the east-west cross section and may be continuous in the north-south extent. The aquitard lies approximately 26.5 to 29.5 feet bls at the pumping wells and ranges in thickness from 3 to 13.5 feet.

## F.4.5.3 Site-Specific Hydrogeology

In the vicinity of Pump Test 8, the A1-aquifer zone consists of interfingering sands and gravels as shown in Figures F4.5-3 and F4.5-4. Water levels in A1- and A2-aquifer zone monitoring wells are above the top of these units suggesting confined conditions; however, due to the presence of many interbeds of clays and sands, the aquifer system is considered to be semiconfined.

Groundwater elevations were calculated from static water level measurements obtained prior to the constant discharge pump test. Figures F4.5-5 and F4.5-6 are potentiometric surface maps of the A1- and A2-aquifer zones, respectively. Local groundwater flow in the A1-aquifer zone is northeasterly in the vicinity of the pumping well. The approximate horizontal gradient is 0.004. Groundwater flow in the A2-aquifer zone is also northeasterly with an approximate horizontal gradient of 0.005.

## F.4.5.4 Overview of Testing

## F.4.5.4.1 A1-Aquifer Zone

A step-drawdown test was conducted on pumping well W09-35(A1) on September 26, 1991 for 4 hours. The step-drawdown test was used to establish an optimal pump rate for the A1-aquifer zone constant discharge pump test. See Section F.4.5.5.1 for a complete discussion of the step-drawdown test.

A slug test was performed on the aquitard piezometer PZ9.8-1(AQ) to determine the hydraulic conductivity of the A1/A2 aquitard. Slug tests were conducted at two other Site 9 locations to investigate the hydraulic properties of the aquitard (refer to Sections F.4.2.7 and F.4.3.7).

The A1-aquifer zone pump test began on November 14, 1991. Pumping well W09-35(A1) was pumped at 4.7 gpm for approximately 10 hours, 30 minutes. The test site was also monitored for a 12-hour recovery period. The pumping well and 7 monitoring wells were monitored with a data logger, and another 11 monitoring wells were monitored with calibrated water level probes. Table F4.5-1 summarizes the pump test results for the wells and piezometers observed during Pump Test 8. Monitoring well and piezometer locations are shown relative to other Site 9 features in Figure F4.5-1. Quantitative analyses were performed on the pumped well and on piezometers PZ9.8-2(A1), PZ9.8-4(A1), and PZ9.8-6(A1). Analytical methods and results are summarized in Table F4.5-2. The remaining monitoring

wells and piezometers were monitored to provide qualitative information on the degree of interconnection between the A1- and A2-aquifer zones and on possible heterogeneities within the A1- and A2-aquifer zones. No drawdown was observed in the remaining monitoring wells.

## F.4.5.4.2 A2-Aquifer Zone

A step-drawdown test was conducted on well W09-20(A2) on September 27, 1991 for 4 hours, 46 minutes. Results of the test were used to establish an optimum pumping rate for the A2-aquifer zone pump test. See Section F.4.5.5.2 for a complete discussion of the step-drawdown test.

The A2-aquifer pump test began on November 19, 1991. Well W09-20(A2) was pumped at 14 gpm for approximately 24 hours. The test site was also monitored for a 19-hour recovery period. The pumping well and 7 monitoring wells were monitored with a data logger, and another 11 monitoring wells were monitored with calibrated water level probes (Table F4.5-1). Monitoring well and piezometer locations were shown relative to other Site 9 features in Figure F4.5-2. Quantitative analyses were performed on the pumped well and on piezometers PZ9.8-3(A2), PZ9.8-5(A2), PZ9.8-7(A2), and PZ9.8-1(AQ). Analytical methods and results are summarized in Table F4.5-2. The remaining monitoring wells and piezometers were monitored to provide qualitative information on the degree of interconnection between the A1- and A2-aquifer zones and on possible heterogeneities within the A1- and A2-aquifer zones.

## F.4.5.5 Step-Drawdown Test

## F.4.5.5.1 A1-Aquifer Zone

A step-drawdown test was conducted September 26, 1991 at pumping well W09-35(A1) to determine the optimum pumping rate for the 24-hour constant discharge test.

The step-drawdown test consisted of three steps of pumping at successively higher discharge rates of 1.1, 2.4, and 5.7 gpm. The steps ranged from 30 minutes to 2 hours, 18 minutes with a total test time of 4 hours. The first two steps were pumped until drawdown appeared stabilized. A graph and table of the discharge rate and drawdown for each step are shown in Figure F4.5-7. The drawdown resulting from the three steps ranged from 0.95 to 5.7 feet with a total drawdown of approximately 7.5 feet at the end of the test. During the third step,

drawdown did not stabilize. From review of the time-drawdown data and the specific capacities completed for the first two steps, a discharge rate of 4.7 gpm is considered optimal.

## F.4.5.5.2 A2-Aquifer Zone

The step-drawdown test was conducted on September 27, 1991 at pumping well W09-20(A2) and consisted of five steps of pumping at successively higher discharge rates of 1.4, 3.0, 6.1, 12.0, and 23.0 gpm. The steps ranged from 28 minutes to 1 hour, 20 minutes with a total test time of 4 hours, 46 minutes. Except for Step 5 (23 gpm), each step was pumped until drawdown appeared to have stabilized (Figure F4.5-8). The drawdown resulting from the five steps of this test ranged from 0.70 foot to greater than 20.1 feet with a total drawdown of more than 20.8 feet. Drawdown data for the fifth step could not be quantitatively used because stabilization for this step was not reached. The pumping well capacity appears to be between 12 and 23 gpm. A rate of 14 gpm was chosen for the long-term aquifer test.

## F.4.5.6 Aquifer Analysis

## F.4.5.6.1 A1-Aquifer Zone

Transmissivity was estimated at the pumping well using the Theis recovery method (Figure F4.5-9) and Cooper-Jacob Method (Figure F4.5-10). Transmissivity and storativity were estimated from data obtained from three piezometers screened in the A1-aquifer zone using the Theis method (Figures F4.5-11, F4.5-13, and F4.5-15) and the Hantush-Jacob method for semiconfined conditions (Figures F4.5-12, F4.5-14, and F.4.5-16). Results are presented in Table F4.5-2. Based on site stratigraphy and the character of the observed data, the A1-aquifer zone is considered semiconfined; therefore, results of the Hantush-Jacob analysis are considered representative of A1-aquifer zone conditions. Results of the Theis method are provided for comparison.

Transmissivity was also estimated at the pumping well using the Theis recovery method (Figure F4.5-9) and the Cooper-Jacob Method (Figure F4.5-10). The recovery data are very strongly displaced toward the left of the theoretical position of the curves. This effect suggests that recovery is incomplete and is consistent with lateral pinching out of sand zones as suggested by analysis of the drawdown curves. The evaluation of the pumping data confirms that the transmissivity obtained from the recovery data is representative of the aquifer.

The range of transmissivities at the A1 piezometers investigated during Pump Test 8 ranged from  $0.074 \text{ ft}^2/\text{min}$  (S =  $5.1 \times 10^{-3}$ ) in piezometer PZ9.8-2(A1) to  $0.74 \text{ ft}^2/\text{min}$  (S =  $4.0 \times 10^{-3}$ ) in piezometer PZ9.8-4(A1). Using an estimated average thickness of 8 feet, the corresponding range of hydraulic conductivity is 0.0093 to 0.093 ft/min. Transmissivity at the pumping well was estimated to be  $0.541 \text{ ft}^2/\text{min}$  (Table F4.5-2).

Semiconfined curves matched the early time data well. The values of r/B provided on the curve match plots of the Hantush semiconfined curves (Figures F4.5-12, F4.5-14, and F4.5-16) indicate aquitard leakage. Because the time-drawdown plots were closely matched to the type curves that neglect storage in the aquitard, the leakage may have passed through the aquitard from the underlying aquifer. As discussed in Section F.4.5, a vertical hydraulic gradient exists from the A2-aquifer zone to the A1-aquifer zone.

An important aspect of the pump test results for the A1-aquifer zone pump test is the existence of a possible limiting boundary affecting the A1-aquifer zone. The drawdown trend in the pumping well exhibits a flattening of the response curve followed by a sharp positive deviation from approximately 4 hours after pumping began. This trend continued until the water level encroached on the well screen 10 hours after the test began.

The following are two possible causes for the observed A1-aquifer zone boundary effect:

- Low permeability boundary to horizontal flow exists between the pumping well and the A1 monitoring wells and piezometers.
- Vertical stratification may exist near the pumping well screen interval.

Both of these conditions would cause decreasing yield with time resulting in an increased rate of drawdown.

Two background monitoring wells were used to monitor background conditions during the pumping and recovery phases of the A1 pump test. A1-aquifer zone monitoring well W09-37(A1), located approximately 615 feet south of the pumping well (Figure F4.5-1), exhibited a 0.01-foot decline in water level during the pumping phase of the test with a 0.03-foot decline during the recovery phase. A2-aquifer zone monitoring well W09-17(A2), located approximately 500 feet south of the pumped well, behaved similarly with a 0.02-foot decline in water level during pumping followed by an additional decline of 0.01 foot during the

recovery phase. As shown in Figure F4.5-16, the three piezometers used to collect data for the quantitative analysis were located within 55 feet of the pumped well and experienced drawdowns greater than 0.3 foot. A site-wide change in water level appears to have affected the A1- and A2-aquifer zones during the pump test. Although the magnitude of this change may be up to 10 percent of the total water level change during the pump test, its effect is considered negligible.

The pump test influenced A1-aquifer zone piezometers at a maximum distance of 53.5 feet by the end of the 10-hour test (Figure F4.5-17). The short duration of the test and the low discharge rate precluded the development of a substantial area of influence (Figure F4.5-1).

## F.4.5.6.2 A2-Aquifer Zone

Values of transmissivity and storativity were calculated from data obtained at five wells or piezometers (Table F4.5-2). The deepest piezometers were analyzed using the Theis method and the Hantush-Jacob method for semiconfined conditions (Figures F4.5-17 through F4.5-24). Based on site stratigraphy and the character of the observed data, the A2-aquifer zone is considered semiconfined; therefore, results of the Hantush-Jacob analysis are considered representative of A2-aquifer zone conditions. Results of the Theis method are provided for comparison.

The pumping well was analyzed using the Theis recovery method (Figure F4.5-16). Recovery data define a line that appears to be strongly shifted to the right of the theoretical position of the recovery curve. This shift suggests that the aquifer has received recharge from an adjacent layer.

The range of estimated transmissivities at the A2-aquifer zone piezometers varied from  $0.11 \, \mathrm{ft^2/min}$  (S =  $2.3 \times 10^{-3}$ ) in piezometer PZ9.8-1(AQ) to  $0.33 \, \mathrm{ft^2/min}$  (S = 0.066) in piezometer PZ9.8-3(A2). Using an estimated average thickness of 10 feet, the corresponding range of hydraulic conductivity is  $0.011 \, \mathrm{to} \, 0.033 \, \mathrm{ft/min}$ . Transmissivity at the pumping well was estimated to be  $0.47 \, \mathrm{ft^2/min}$  (Table F4.5-2). The piezometer designated as the aquitard piezometer experienced drawdown during pumping, and, therefore does not appear representative of the aquitard material. Alternatively, the observed drawdown may indicate that the A1/A2 aquitard in this vicinity allows interconnection between the A1- and A2-aquifer zones.

The values of r/B provided on the curve match plots of the Hantush semiconfined curves (Figures F4.5-18 through F4.5-24) indicate aquitard leakage. Because the time-drawdown plots were closely matched to the type curves that neglect storage in the aquitard, the leakage can be reasonably assumed to have passed through the aquitard from an overlying (or underlying) aquifer. Further, the transmissivity estimated from piezometer PZ9.8-1(AQ) is the same order of magnitude as other A2-aquifer zone transmissivity estimates. This implies that the A1/A2 aquitard may not act as a barrier to groundwater flow in this area.

Background water levels were monitored in monitoring well W09-37(A1) located 615 feet from the pumping well (Figure F4.5-2). The water level in the A1-aquifer zone fell 0.02 foot during pumping and rose 0.02 foot during recovery. This may indicate an effect due to pumping in the A2-aquifer zone rather than one caused by site-wide water level changes. As illustrated in Figure F4.5-25, the four piezometers involved in the quantitative analysis were located within 50 feet of the A2-aquifer zone pumping well and exhibited water level changes greater than 1 foot during the pump test. The relatively small water level changes observed in the background well are therefore considered to have negligible effects on the results of quantitative analysis.

The pump test influenced wells at a maximum distance of 476 feet from the pumping well by the end of the 24-hour test. Some site wells responded with more drawdown than those wells closer to the pump (Figure F4.5-25). Also, the resulting shape of the area of influence is irregular (Figure F4.5-2). This is most likely the result of variations in aquifer geometry and hydraulic properties.

The pre-pumping test vertical hydraulic head between the A1- and A2-aquifer zones was 0.08 foot between wells W09-20(A2) and W09-35(A1) (Table F4.5-3). This head difference indicates the potential for flow from the A2- to the A1-aquifer zones.

## F.4.5.7 Slug Test Analysis

A slug test was performed on the piezometer PZ9.8-1(AQ). The reported transmissivity, storativity, and hydraulic conductivity values are probably not representative of aquitard characteristics as noted previously (Sections F.4.2.7 and F.4.3.7). The method of Cooper, et al. (1967) was used to analyze the slug test data. The calculated value of transmissivity was  $1.1 \times 10^{-2}$  ft<sup>2</sup>/min. Storativity was  $1 \times 10^{-6}$ . Assuming an aquitard thickness of 11 feet, the hydraulic conductivity is  $1.0 \times 10^{-3}$  ft/min (Figure F4.5-26).

## F.4.5.8 Groundwater Quality

## F.4.5.8.1 A1-Aquifer Zone

The A1 pump test ended prematurely 2 hours after the first 8-hour sample, and results are inconclusive (Table F4.5-4). TCE was the most prominent compound detected with a concentration of 3,100 ppb.

#### F.4.5.8.2 A2-Aquifer Zone

Three rounds of sampling were conducted during the A2 pump test. Results indicate that pumping created no significant change in contaminant levels. Aquifer test sampling results for groundwater TCE concentrations ranged from 12,000 to 14,000 ppb. 1,1-DCE concentrations ranged from 190 to 200 ppb during the test (Table F4.5-5).

# F.5.0 Summary and Conclusions

Pump tests were conducted at Site 8, the Waste Oil Transfer Area, and Site 9, the Old Fuel Farm and Old NEX Auto Service Station, to obtain data necessary for the characterization of the A-aquifer system. Testing was conducted during October and November 1991. Data required for aquifer characterization included specific capacity, transmissivity, figure of the aquifer zones, and radius of influence.

Results of a single pump test at Site 8 indicate that the A1- and A2-aquifer zones make up a single semiconfined zone with estimated transmissivities ranging from 8.19 to 18.14 ft<sup>2</sup>/min. Storativities were typical for semiconfined to confined conditions and ranged from 0.0003 to 0.0069. The estimated average thickness of the aquifer zone is 15 feet. These data are summarized in Table F5-1.

Pumping was conducted at a constant rate of 15 gpm over approximately 24 hours and affected wells to a radial distance of approximately 300 feet. The configuration of the induced cone of depression does not appear affected by major lateral variations in aquifer geometry or hydraulic properties. Localized zones of recharge were not observed. Relatively uniform site-wide recharge from an underlying or overlying zone resulted in the classification of the aquifer as semiconfined; however, the magnitude of this recharge is small, implying that hydraulic connection between the A1- and A2-aquifer zones and adjacent materials is not extensive.

Four pump tests were conducted at Site 9. Results of the tests indicate that the A-aquifer system in this vicinity comprises two interconnected zones, designated A1 and A2. Hydraulic parameters of the A1- and A2-aquifer zones are summarized in Table F5-2.

Estimated transmissivities in the A1-aquifer zone ranged from 0.54 to 29.55 ft<sup>2</sup>/min. Estimated storativities ranged from 0.01 to 0.04 and exceeded typical values for semiconfined conditions. The high estimated storativities may be due to model bias or may imply inelastic aquifer deformation in response to falling pore pressures during pumping. If this is the case, it is anticipated that land subsidence may be an important consideration in future groundwater extraction programs.

Estimated transmissivities in the A2-aquifer zone ranged from 0.29 to 36.81 ft<sup>2</sup>/min. Storativity ranged from 0.0002 to 0.49. The majority of the storativity estimates were near the geometric mean of 0.002, which is typical for semiconfined conditions.

Estimated hydraulic parameters of the A1- and A2-aquifer zones appear to be more closely related to the horizontal coordinate than to the aquifer zone. Both zones exhibited a relatively wide range in aquifer parameters, implying that preferred migration pathways for groundwater contaminants exist in the vicinity of Site 9. Groundwater declines induced in the A1- and A2-aquifer zones during constant discharge pumping exhibited north-south elongation, thus providing further evidence of aquifer heterogeneity.

Analysis of the A1/A2 aquitard at Site 9 is summarized in Table F5-3. Hydraulic conductivities estimated from slug test data and geotechnical testing ranged from 9.1 x 10<sup>-5</sup> to 1.0 x 10<sup>-3</sup> ft/min and provided further evidence of lateral heterogeneity in the A-aquifer system. Further, hydraulic connection between the A1- and A2-aquifer zones is supported by the relatively high hydraulic conductivities estimated at some aquitard test locations.

Chemical analysis of groundwater collected from pumping wells during aquifer testing indicated that concentrations of VOCs are stable over 24-hour measurement periods.

# F.6.0 Bibliography

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# TABLE F4.0-1 Net Drawdowns in Observation Wells and Piezometers for End of Pump Tests, Sites 8 and 9 Moffett Field

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PUMP TEST 7 -	CALCULAT	ED DRAWDO	WN DATA FO	OR PUMPING						<del></del>	<u></u>
WELL	W08-06	PZ8.7-1	W08-02	PZ8.7-2	W08-01	WO8-12	W08-10	W08-05	W08-11	W08-04	MEW-92
WELL	(A1-A2)	(A-1)	(A-2)	(A-1)	(A-1)	(A-2)	(A-1)	(A-1)	(A-2)	(A-1)	(A-1)
Distance (ft)	0	45.3	46.6	80	140	200	153	173	277	287	350
Drawdown (ft)	7.601	0.609	0.536	0.449	0.205	0.291	0.362	0.158	0.18	0.17	0.14
Drawdown is tak	en from 1375	minutes.									
PUMP TEST 1 -	CALCULAT	ED DRAWDO	WN DATA FO	OR PUMPING							<del></del>
WELL	W09-09	W56-01	FP9-2	PZ 9.1-1	PZ 9.1-4	PZ 9.1-2	PZ9.1-3	W09-47	W56-02	W09-02	W09-13
	(A-2)	(A-1)		(AQ)	(A-1)	(A-2)	(A-2)	(A-1)	(A-1)	(A-1)	(A-2)
Distance (ft)	0	7.5	8	25.4	25.4	25.4	65.5	106	115	130.5	139.3
Drawdown (ft)	7.07	1.101	1.045	2.31	1.823	3.139	1.75	0.618	0.82	0.91	1.62
Drawdown is tak	en from 1,405	5 minutes.				_	-				
PUMP TEST 3 -	CALCULAT	ED DRAWDO	WN DATA FO	OR PUMPING							
WELL	W09-22	PZ 9.3-1	PZ 9.3-2	PZ 9.3-3	PZ 9.3-4	PZ 9.3-5	W29-03	W29-09	FP9-1	W29-08	W29-02
	(A-2)	(AQ)	(A-1)	(A-2)	(A-1)	(A-2)	(A-1)	(A-2)		(A-2)	(A-1)
Distance (ft)	0	14.3	14.3	14.30	25	25	77.5	. 68	73	111	105
Drawdown (ft)	5.235	0.97	0.862	3.86	0.827	3.859	0.43	3.044	0.75	1.08	0.36

**TABLE F4.0-1** 

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WELL	W9-6	W9-1	W09-27	W29-06	W09-31	W09-26	W09-28	W61-01	W29-05	W09-35	
	(A-1)	(A-1)	(A-2)	(A-1)	(A-1)	(A-1)	(A-2)	(A-1)	(A-1)	(A-1)	
Distance (ft)	197	230	300	205	213	362	383	270	356	650	
Drawdown (ft)	0.23	0.18	0.12	0.3	0.35	0.03	0.2	0.07	0.07	0	
Drawdown is tak	en from 1,375	minutes.	<u> </u>				· · · · · · · · · · · · · · · · · · ·				
PUMP TEST 5(A	A1) - CALCU	LATED DRAV	WDOWN DAT	A FOR PUMP	ING						
WELL	W09-38	W09-41	PZ9.5-6	PZ 9.5-7	PZ 9.5-1	PZ 9.5-4	PZ9.5-5	PT9-2	PT9-3	W09-16	W09-19
	(A-1)	(A-2)	(A-1)	(A-2)	(AQ)	(A-1)	(A-2)	(A-1)	(A-2)	(A-1)	(A-1)
Distance (ft)	0	15	36.7	34.7	43.7	46.7	41	223	223	240	303
Drawdown (ft)	7.073	1.13	1.604	0.937	1.034	1.319	0.506	0.97	0.36	0.54	0.7
W.C.I.	W09-37	11/00 17	MENU 46	N100 26							
WELL	(A-1)	W09-17 (A-2)	MEW 46 (A1-A2)	W09-36 (A-2)							
Distance (ft)	292	383	421	(A-2)							
Drawdown (ft)	0.06	0.09	0.16	0.14			·				
Drawdown is tak	<del></del>		0.10	0.14					<u></u>		· · · · · · · · · · · · · · · · · · ·
. <del></del>			UDOWN DAT	A FOR BUILD		<del></del>		<del></del>			
PUMP TEST 5(A	·				T			<u> </u>			
WELL	W09-41	W09-38	PZ9.5-6	PZ 9.5-7	PZ 9.5-1	PZ 9.5-4	PZ9.5-5	PT 9-2	PT 9-3	W09-37	W09-16
	(A-2)	(A-1)	(A-1)	(A-2)	(AQ)	(A-1)	(A-2)	(A-1)	(A-2)	(A-1)	(A-1)
Distance (ft)	0	15	21.9	19.9	53.7	56.7	51.7	208	208	292	234
Drawdown (ft)	6.229	0.142	0.256	1.707	1.359	0.202	0.721	0.12	0.13	0.01	0.07

#### TABLE F4.0-1

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WELL (%)	W09-19 (A-1)	W09-17	W09-36	W09-14						1	
D: (C)	(A-1)		<del></del>	W03-14	W09-18	MW09-44	W09-08	W09-40			<u></u>
D: (6)		(A-2)	(A-2)	(A-2)	(A-1)	(A-1)	(A-2)	(B-2)			
Distance (ft)	288	380	472	420	574	492	500	508			
Drawdown (ft)	0.08	0.03	0	0.02	0.01	0.07	0.03	0.02			
Drawdown is taken from 1480 minutes											
PUMP TEST 8(A1) - CALCULATED DRAWDOWN DATA FOR PUMPING											
	W09-35	W09-20	PZ 9.8-1	PZ 9.8-2	PZ 9.8-3	PZ 9.8-5	PZ9.8-4	PZ9.8-6	PZ9.8-7	W09-15	
TIME	(A-1)	(A-2)	(AQ)	(A-1)	(A-2)	(A-2)	(A-1)	(A-1)	(A-2)	(B-2)	
Distance (ft)		5	12.2	12.2			30.2	53.5			
Drawdown (ft)	14.32	0.333	0.368	0.944	0.217	0.056	0.331	0.297	0.26	0.03	
Drawdown is taken from 620 minutes											
PUMP TEST 8(A2)	) - CALCUI	LATED DRAV	VDOWN DAT	A FOR PUMP	ING						
WELL	W09-20	W09-35	PZ 9.8-1	PZ 9.8-2	PZ 9.8-3	PZ 9.8-4	PZ9.8-5	PZ9.8-7	PZ9.8-6	W09-15	
	(A-2)	(A-1)	(AQ)	(A-1)	(A-2)	(A-1)	(A-2)	(A-2)	(A-1)	(B-2)	
Distance (ft)	0	5	12.2	12.2	12.2	30.2	30.2	48.5	48.5	92	-
Drawdown (ft)	17.159	1.056	2.681	1.007	1.303	0.183	1.854	1.859	0.44	0.15	
WELL	W09-21	W09-28	W09-36	W09-33	W09-14	W09-18	W09-17	W09-23	W09-37	_	
	(A-2)	(A-2)	(A-2)	(A-2)	(A-2)	(A-1)	(A-2)	(A-1)	(A-1)		
Distance (ft)	243	334	336	305	346	387	476	321	582		
Drawdown (ft)	0.15	0.17	0.08	0.09	0.27	0.07	0.08	0.06	0.02		
Drawdown is taken	from 1315	minutes									

### TABLE F4.1-1 DESCRIPTION OF WELLS AND PIEZOMETERS PUMP TEST 7, SITE 8 MOFFETT FIELD

Well Designation	Well	Screened Interval (ft)	Filter Pack Interval (ft)	Casing Diam.(in)	Distance From Pumping Well (ft)	Pumping Influence	Comments
Pumping Well	W08-6(A1-A2)	17 to 37	15 to 40	4	N/A	Y	Monitor w/ transducer
Piezometer	PZ 8.7-1(A1)	29.5 to 44.5	27 to 45	2	45.3	<del></del>	Monitor w/ transducer
Piezometer	PZ 8.7-2(A1)	29.5 to 44.5	27.5 to 45	2	80	Y	Monitor w/ transducer
					·		
Monitoring Well	MEW-92(A1)	18 to 33	16 to 35	4	350	Y	Monitor w/ W.L. meter
Monitoring Well	MEW-82(B2)	71 to 86	67 to 88	4	350	N	Monitor w/ W.L. meter
Monitoring Well	W08-01(A1)	19.8 to 30	18 to 30	2	140	Y	Monitor w/ transducer
Monitoring Well	W08-02(A2)	43 to 48	39 to 50.5	4	46.6	Y	Monitor w/ transducer
Monitoring Well	W08-04(A1)	17 to 22	15.3 to 22.5	4	287	Y	Monitor w/ W.L. meter
Monitoring Well	W08-05(A1)	21.5 to 26.5	18 to 28.5	4	173	Y	Monitor w/ transducer
Monitoring Well	W08-08(A1)	16 to 26	14 to 27	4	610	N	Monitor w/ W.L. meter
Monitoring Well	W08-10(A1)	24 to 34	22 to 37	4	153	Y	Monitor w/ transducer
Monitoring Well	W08-11(A2)	28 to 38	26 to 40	4	277	Υ	Monitor w/ W.L. meter
Monitoring Well	W08-12(A1)	23 to 33	21 to 40	4	200	Y	Monitor w/ transducer

Notes:

N/A: Not Applicable PZ: Piezometer

W.L.: Water Level meter

MEW: Middlefield-Ellis-Wisman RI/FS Well

Table F4.1-2

Aquifer Classification and Pump Test Analytical Results
Pump Test 7, Site 8

Moffett Field

Well Number	Distance From Pump Aquifer Classification		Analytical Method	Transmissivity (ft <sup>2</sup> /min)	Storativity
W08-06(A2-A2)	0	Semiconfined	Theis Recovery	2.16	N/A
PZ8.7-1(A1)	44.3	Semiconfined	Cooper-Jacob Mod. Hantush Leaky Storage)	2.50 1.42	1.9 x 10 <sup>-4</sup> 6.2 x 10 <sup>-4</sup>
PZ8.7-2(A1)	80	Semiconfined	Cooper-Jacob Mod. Hantush-Leaky (No Storage)	2.67 2.43	6.6 x 10 <sup>-4</sup> 9.2 x 10 <sup>-4</sup>
W08-10(A1)	153	Semiconfined	Hantush Leaky (Storage)	1.24	3.8 x 10 <sup>-5</sup>
W08-12(A1-A2)	200	Semiconfined	Hantush Leaky (Storage)	1.00	1.1 x 10 <sup>-4</sup>

### TABLE F4.1-3 A1/A2 AQUIFER ZONE HEAD DIFFERENTIALS PUMP TEST 7, SITE 8 MOFFETT FIELD

Well Pairs	Vertical Head Elevation of Difference (ft.) Head (ft, msl)		Notes
W8-04(A1), W8-11(A2)	0.78	-1.64, -2.42	vertical flow from A1 to A2 vertical flow from A1 to A2
W8-06(A1-A2), W8-02(A2)	0.21	-1.88, -2.09	

CALZSR3AMOPZ.WIGAj

#### **Table F4.1-4 Groundwater Analytical Results** Pump Test 7, Site 8 **Moffett Field**

WELL No. W08-06 (A1)

Date Sample Taken Time Samples Taken			4/24/91 Quarterly Sampling 2Q91	11/25/91 23:05 7.2 Hours*	11/26/91 08:15 16.4 Hours	1.	26/91 5:00 Hours
COMPOUND	CRQL	UNITs					
1,1,1-Trichloroethane 1,1-Dichloroethane	10 10	ug/L° ug/L	45 12	27 10	24 10	J	21 8
1,1-Dichloroethene Methylene Chloride Trichloroethene	10 10 10	ug/L ug/L ug/L	12	10 J <sup>d</sup> 7	J 4	] ]	7 10 2

NOTES: a Pump test began at 1553 on 11/25/91.

b CRQL = Contract Required Quantitation Limit.
c ug/L = parts per billion (ppb).
d J = Estimated value.

### TABLE F4.2-1 DESCRIPTION OF WELLS AND PIEZOMETERS PUMP TEST 1, SITE 9 MOFFETT FIELD

Well		Screened	Filter Pack	Casing	Distance From	Pumping	
Designation	Well	Interval (ft)	Interval (ft)	Diam.(in)	Pumping Well	Influence	Comments
Pumping Well	W09-09(A2)	35' TO 45'	33.5 to 46	4	N/A		Monitor w/ transducer
			<del></del>				·
Piezometer	PZ9.1-1(AQ)	28.5 to 29.5	28.4 to 29.5	2	25.4	Y	Monitor w/ transducer
Piezometer	PZ9.1-2(A2)	32.7 to 47.7	31 to 49	2	25.4	Y	Monitor w/ transducer
Piezometer	PZ9.1-3(A2)	30 to 45	28.5 to 45	2	65.5	Y	Monitor w/ transducer
Piezometer	PZ9.1-4(A1)	15.5 to 25.5	14.2 to 25.5	2	25.4	<u> </u>	Monitor w/ transducer
Monioring Well	FP9-2	7 to 22	6 to 23	4	8	Y	Monitor w/ transducer
Monitoring Well	W09-47(A1)			4	106	Υ	Monitor w/ transducer
Monitoring Well	W56-01(A1)	10.5 to 25.5	7.2 to 26	4	7.5	Y	Monitor w/ transducer
Monitoring Well	W56-02(A1)	10.5 to 24.5	7 to 25.5	4	115	Y	Monitor w/ W.L. meter
Monitoring Well	W09-01(A1)	19.8 to 30	18 to 30	2	206	Y	Monitor w/ W.L. meter
Monitoring Well	W09-02(A1)	20.8 to 31	18 to 31	2	130.5	Y	Monitor w/ W.L. meter
Monitoring Well	W09-06(A1)	15.2 to 25.2	12.8 to 26.5	4	240	Y	Monitor w/ W.L. meter
Monitoring Well	W09-07(A1)	13.3 to 33.3	10 to 35	4	131.2	Y	Monitor w/ W.L. meter
Monitoring Well	W09-13(A2)	38 to 43	36 to 46	4	139.3	Y	Monitor w/ W.L. meter
Monitoring Well	W09-25(A2)	29.5 to 39.5	27.5 to 42	4	260	Y	Monitor w/ W.L. meter
Monitoring Well	W09-31(A1)	21 to 26	19 to 27	4	538	N	Monitor w/ W.L. meter
Monitoring Well	W09-34(A2)	27 to 42	25.5 to 44.6	4	353	Υ	Monitor w/ W.L. meter
Monitoring Well	W09-39(B2)	86 to 96	82 to 97	4	270	Y	Monitor w/ W.L. meter
Monitoring Well	W09-22(A2)	37 to 47	35 to 50	4	437	Ý	Monitor w/ W.L. meter
Monitoring Well	* W09-28(A2)	38 to 48	36 to 50	4	692	N	Monitor w/ W.L. meter
Monitoring Well	* W09-20(A2)	30 to 45	28 to 46.5	4	785	N	Monitor w/ W.L. meter

Notes:

N/A: Not Applicable

FP: Free Product Well

PZ: Piezometer

W.L.: Water Level Meter

Table F4.2-2

Aquifer Classification and Pump Test Analytical Results
Pump Test 1, Site 9
Moffett Field

Well Number	Distance Well Number From Pump		Analytical Method	Transmissivity (ft <sup>2</sup> /min)	Storativity	
W09-09(A2)	0	Semiconfined (Pumping data)	Theis Recovery Neuman Partial Penetration	1.07 0.70	N/A N/A	
PZ9.1-2(A2)	25.4	Semiconfined	Theis Hantush Leaky (No Storage)	0.71 0.75	1.2 x 10 <sup>-4</sup> 1.2 x 10 <sup>-4</sup>	
PZ9.1-3(A2)	65.6	Semiconfined	Theis Hantush-Leaky (No Storage)	1.52 1.64	7.2 x 10 <sup>-5</sup> 7.1 x 10 <sup>-5</sup>	
W09-13(A2)	139.3	Semiconfined	Theis Hantush Leaky (No Storage)	3.31 2.74	4.0 x 10 <sup>-6</sup> 1.1 x 10 <sup>-5</sup>	
W09-25(A2)	260	Semiconfined	Theis Hantush Leaky (No Storage)	5.0 4.8	3.5 x 10 <sup>-4</sup> 3.7 x 10 <sup>-4</sup>	

## TABLE F4.2-3 A1/A2 AQUIFER ZONE HEAD DIFFERENTIALS PUMP TEST 1, SITE 9 MOFFETT FIELD

Well Pairs	Vertical Head Difference (ft.)	Elevation of Head (ft, msl)	Notes		
		<del>-</del>			
  W9-07 (A1), W9-13(A2)	0.19	10.13,10.32	vertical flow from A2 to A1		
FP9-02, W9-09(A2)	0.89	8.84,9.73	vertical flow from A2 to A1		

C:123R3MOF1.WKVj

### Table F4.2-4 Neuman-Witherspoon Ratio Method for PZ9.1-1(AQ) Pump Test 1, Site 9 NAS Moffett Field

Terms:

Pumped Aquifer = A2

s = drawdown observed (pumped aquifer)(ft)

sc = drawdown observed (aquitard) (ft)

r = distance of PZ9.1-1(aq)from pump = 25.5 feet

t = time in minutes

T = transmissivity = 4.36 ft <sup>2</sup> min(pumped aquifer)

S = storativity in A2 = 3.4E - 03

u\_=Neuman-Witherspoon solution for aquitards

u = Theis solution for aquifers

z = distance from aquitard piezometer screen to top of A2= 1.5 feet

K' = vertical hydraulic conductivity of aquitard

Ss' = specific storage of aquitard, from laboratory testing = 6.07E-04

	1 min	ute (t1)	2 minu	ites (t2)	4 minu	tes (t3)	6 minu	tes (t4)	
	s 1.267	sc 0.063	s 1.666	sc 0.079	s 1.958	sc 0.193	s 2.106	sc 0.294	
$sc/s = W(u,u_c)/W(u)$	0.0497		0.0	0.0474		0.0986		0.1396	
$u = r^{2S/4Tt}$	1.268E-01		6.338	E-02	3.169E-02		2.113E-02		
1/u <sub>c</sub> *	0.82		0.71		1.00		1.40		
K'/Ss'**	0.461		0.199		0.141		0,131		

Mean K'/Ss' = 0.233

Vertical Hydraulic Conductivity for the aquitard, K' = 1.4E-04 ft/min

<sup>\*-1/</sup>u is obtained using Neuman-Witherspoon's nomogram

<sup>\*\*-</sup> K'/Ss' =  $1/u_{c}$  \* $z^{2/4t}$ 

#### **Table F4.2-5 Groundwater Analytical Results** Pump Test 1, Site 9 **Moffett Field**

WELL No. W09-09 (A2)

Date Sample Taken Time Samples Taken			Quart	5/16/91 erly Sampling 2Q91		1/23/91 14:00 75 Hours*	11/23/91 22:00 25.75 Hours	
COMPOUND	CRQL <sup>b</sup>	UNITs						
1,1,1-Trichloroethane	10	ug/L°	J <sup>d</sup>	. 22	J	13		
1,1-Dichloroethane	10	ug/L	J	33	<b>J</b>	25		
1,1-Dichloroethene	10	ug/L	J	49	J	24	24	
1,2-Dichloroethene (Total)	10	ug/L		63	J	56	60	
Methylene Chloride	10	ug/L	J	25		230	200	
Trichloroethene	10	ug/L		2,200		1,900	1,800	

NOTES: a Pump test began at 2015 on 11/22/91.

<sup>&</sup>lt;sup>b</sup> CRQL = Contract Required Quantitation Limit.

c ug/L = parts per billion (ppb).
d J = Estimated value.

### TABLE F4.3-1 DESCRIPTION OF WELLS AND PIEZOMETERS PUMP TEST 3, SITE 9 MOFFETT FIELD

Well		Screened	Filter Pack	Casing	Distance From	Pumping	
Designation	Well	Interval (ft)	Interval (ft)	Diam.(in.)	Pumping Well (ft)	Influence	Comments
Pumping Well	W9-22(A2)	37 to 47	35 to 50	4	N/A	Y	Monitor w/ transducer
Discounting	D70.2.1/AO\	28 to 29	27.5 to 29.5		14.3	<del></del> Y	Monitor w/ transducer
Piezometer	PZ9.3-1(AQ)			2	14.3 14.3	Y	Monitor w/ transducer
Piezometer	PZ9.3-2(A1)	15 to 20	14 to 20.5	2		Y	•
Piezometer	PZ9.3-3(A2)	39 to 49	34.5 to 50	2	14.3	Y	Monitor w/ transducer
Piezometer	PZ9.3-4(A1)	15.6 to 20.6		2	25 25	•	Monitor w/ transducer
Piezometer	PA9.3-5(A2)	40 to 50	38 to 51	2	25	Y	Monitor w/ transducer
Monitoring Well	FP9-1	5 to 19.5	4 to 20	4	73	Y	Monitor w/ W.L. meter
Monitoring Well	W09-01(A1)	19.8 to 30	18 to 30	2	230	Y	Monitor w/ W.L. meter
Monitoring Well	W09-06(A1)	15.2 to 25.2	12.8 to 26.5	4	197	Y	Monitor w/ W.L. meter
Monitoring Well	W09-26(A1)	7 to 17	6 to 17.4	4	362	Y	Monitor w/ W.L. meter
Monitoring Well	W09-27(A2)	37.8 to 47.8	35.8 to 49	4	300	Y	Monitor w/ W.L. meter
Monitoring Well	W09-28(A2)	38 to 48	36 to 50	4	383	Y	Monitor w/ W.L. meter
Monitoring Well	W09-31(A1)	21 to 26	19 to 27	4	213	Y	Monitor w/ W.L. meter
Monitoring Well	W09-35(A1)	14 to 24	12 to 25	4	650	N	Monitor w/ W.L. meter
Monitoring Well	W29-02(A1)	7.5 to 17.5	5.3 to 20	4	105	Y	Monitor w/ W.L. meter
Monitoring Well	W29-03(A1)	10.5 to 20.5	7 to 25	4	77.5	Y	Monitor w/ transducer
Monitoring Well	W2905(A1)	10.5 to 20.5	7.5 to 30	4	356	N	Monitor w/ W.L. meter
Monitoring Well	W29-06(A1)	10.5 to 20.5	7 to 30	4	205	Y	Monitor w/ W.L. meter
Monitoring Well	W29-08(A2)	37 to 47	32.6 to 47.5	4	111	Y	Monitor w/ W.L. meter
Monitoring Well	W29-09(A2)	38 to 48	34.5 to 49	4	68	Y	Monitor w/ transducer
Monitoring Well	W61-01(A1)	7.5 to 17.5	5 to 20	4	270	N	Monitor w/ W.L. meter

Notes:

N/A: Not Applicable

FP: Free Product Well

PZ: Piezometer

W.L.: Water Level meter

**Table F4.3-2** 

### Aquifer Classification and Pump Test Analytical Results Pump Test 3, Site 9 Moffett Field

Well Number	Distance Aquifer ell Number From Pump Classification		Analytical Method	Transmissivity (ft <sup>2</sup> /min)	Storativity	
W09-22(A2)	0	Semiconfined	Theis Recovery	0.28	N/A	
PZ9.3-3(A2)	14.3	Semiconfined	Theis Hantush Leaky (No Storage)	0.32 0.32	8.8 x 10 <sup>-5</sup> 8.7 x 10 <sup>-5</sup>	
PZ9.3-5(A2)	25	Semiconfined	Theis Hantush-Leaky (No Storage)	0.30 0.29	6.8 x 10 <sup>-5</sup> 6.8 x 10 <sup>-5</sup>	
W29-08(A2)	111	Semiconfined	Theis Hantush Leaky (No Storage)	0.66 0.53	1.5 x 10 <sup>-3</sup> 1.5 x 10 <sup>-3</sup>	

### TABLE F4.3-3 A1/A2 AQUIFER ZONE HEAD DIFFERENTIALS PUMP TEST 3, SITE 9 MOFFETT FIELD

Well Pairs	Vertical Head Difference (ft.)	Elevation of Head (ft, msl)	Notes
W9-23(A1), W9-28(A2)	0.3	8.34, 8.64	vertical flow from A2 to A1

CALZER A MOPS. W KSA

# Table F4.3-4 Neuman-Witherspoon Ratio Method for PZ9.3 - 1 (AQ) Pump Test 3, Site 9 NAS Moffett Field

Terms:

Pumped Aquifer = A2

s = drawdown observed (pumped aquifer) (ft)

sc = drawdown observed (aquitard) (ft)

r = distance of PZ9.1-1 (aq) from pump = 14.3 feet

t = time in minutes

T = transmissivity = 2.31 ft<sup>2</sup>/min (pumped aquifer)

S = storativity in A2 = 6.84E - 04

u = Neuman-Whitherspoon solution for aquitards

u = Theis solution for aquifers

z = distance from aquitard piezometer screen to top of A2=8.5 feet

K' = vertical hydraulic conductivity of aquitard

Ss' = specific storage of aquitarad, from laboratory testing=1.95E-04

	6 min s 1.86	1		utes (t2)   sc   0.10	24 minu s 2.75	ites (t3)   sc   0.21	
sc/s = W(u,u)/W(u)	0.0	32	0.0	)46	0.0	76	
$u = r^{2S/4Tt}$	2.52	E-03	1.518	E-03	6.316	E-04	
1/u <sub>c</sub> *	0.4	15	0.	52	0.72		
K'/Ss' **	1.35	55	0.9	939	0.5	542 	

Mean K'/Ss' = 0.945

Vertical Hydraulic Conductivity for the aquitard, K' = 1.84E - 04 ft/min

<sup>\* - 1/</sup>uc is obtained using Neuman-Witherspoon's nomogram

<sup>\*\* -</sup> K'/Šs' =  $1/u_s$  \*  $z^{2/41}$ 

#### **Table F4.3-5 Groundwater Analytical Results** Pump Test 3, Site 9 **Moffett Field**

WELL No. W09-22 (A2)

Date Sample Taken Time Samples Taken		5/14/91 Quarterly Sampling 2Q91		11/07/91 22:30 7.9 Hours*		11/08/91 07:00 16.4 Hours		11/08/91 14:00 23.4 Hours		11/08/91 <sup>5</sup> 14:10		
COMPOUND CI	RQL	UNITs										
1,1-Dichloroethane	10	ug/Lª	J.	26			J	26				
1,1-Dichloroethene	10	ug/L	J	55	J	72	J	68	J	58	J	52
1,2-Dichloroethene (Total)	10	ug/L		160	J	160	J	160	J	160	J	140
Methylene Chloride	10	ug/L	J	40	J	210	J	110	J	110	J	250
Trichloroethene	10	ug/L		3,300		3,900		3,600		3,200		2,900

NOTES: <sup>a</sup> Pump test began at 1435 on 11/07/91.
<sup>b</sup> Field Duplicate.
<sup>c</sup> CRQL = Contract Required Quantitation Limit.

d ug/L = parts per billion (ppb).

J = Estimated value.

### TABLE F4.4-1 DESCRIPTION OF WELLS AND PIEZOMETERS PUMP TEST 5, SITE 9 MOFFETT FIELD

Well		Screened	Filter Pack	Casing	Distance I	-rom	Pum	ping	
Designation	Well	Interval (ft)	Interval (ft)	Diam.(in)	Pumping \	Vells(ft)	Influe	ence	Comments
					A1 Pump	A2 Pump	A1	A2	
Pumping Well	W09-38(A1)	12 to 22	9 to 23	4	N/A	15	Y	Y	Monitor w/ transducer
Pumping Well	W09-41(A2)	34 to 44	32 to 46	4	15	N/A	Y	Υ	Monitor w/ transducer
Piezometer	PT9-2(A1)	12.6 to 27.6	12.6 to 30	4	223	208	Υ	Y	Monitor w/ W.L. meter
Piezometer	PT9-3(A2)	36.5 to 51.5	35.5 to 52	4	223	208	Y	Y	Monitor w/ W.L. meter
Piezometer	PZ9.5-1(AQ)	41.5 to 42.5	41.2 to 42.5	4	43.7	53.7	Υ	Y	Monitor w/ transducer
Piezometer	PZ9.5-4(A1)	15 to 30	13 to 30	4	46.7	56.7	Υ	Υ	Monitor w/ transducer
Piezometer	PZ9.5-5(A2)	50 to 55	49 to 57	4	41	51.7	Υ	Y	Monitor w/ transducer
Piezometer	PZ9.5-6(A1)	13.2 to 28.2	11 to 30	4	36.7	21.9	Υ	Y	Monitor w/ transducer
Piezometer	PZ9.5-7(A2)	34 to 44	31.9 to 47	4	34.7	19.9	Y	<u>Y</u>	Monitor w/ transducer
Monitoring Well	W09-08(A2)	33 to 39.5	29 to 40	4	515	500	Y	N	Monitor w/ W.L. meter
_	• •	39 to 49	36.8 to 52	4	429	420	N	N	Monitor w/ W.L. meter
Monitoring Well	W09-14(A2) W09-16(A1)	19 to 29	17 to 30	7	240	234	Y	Y	Monitor w/ W.L. meter
Monitoring Well Monitoring Well	W09-17(A2)	33 to 38	31 to 40	4	383	380	N	N	Monitor w/ W.L. meter
Monitoring Well	W09-18(A1)	14 to 24	12 to 25	4	578	574	N	N	Monitor w/ W.L. meter
Monitoring Well	W09-19(A1)	20 to 30	18 to 32	4	303	288	Y	Y	Monitor w/ W.L. meter
Monitoring Well	W09-36(A2)	33 to 43	28 to 44	7 A	484	472	Ÿ	Ņ	Monitor w/ W.L. meter
Monitoring Well	W09-37(A1)	10 to 20	8 to 21.5	4	292	292	N	N	Monitor w/ W.L. meter
Monitoring Well	W09-40(B2)	90 to 105	88 to 107	<b>7</b> <b>∆</b>	523	508	N	N	Monitor w/ W.L. meter
. •	MEW-81 (A1)	13 to 23	11 to 25	7 A	421	421	N		Monitor w/ W.L. meter
Monitoring Well	• •			7	421	421	Y		Monitor w/ W.L. meter
Monitoring Well	MEW-46(A1)	14 to 34	14 to 34	4		510	N		Monitor w/ W.L. meter
Monitoring Well	W09-46(A1)	17 to 27	15 to 27.5	4	510 750			•	·
Monitoring Well	W09-20(A2)	30 to 45	28 to 47	4	753	743	N	N/A	
Monitoring Well	W09-35(A1)	14 to 24	12 to 25	4	785	775	N		Monitor w/ W.L. meter
Monitoring Well	MW009-44(A1)	15 to 26	11 to 26	4	507	492	<u>Y</u>	<u>Y</u>	Monitor w/ W.L. meter

Notes:

N/A: Not Applicable

PT: Pump Test Well

MEW: Middlefield-Ellis-Wisman RI/FS Well

PZ: Piezometer

W.L.: Water Level meter

Table F4.4-2

Aquifer Classification and Pump Test Analytical Results
Pump Test 5, Site 9
Moffett Field

Well Number	Distance From Pump	Aquifer Classification	Analytical Method	Transmissivity (ft <sup>2</sup> /min)	Storativity
		Α	1 Aquifer Test		
W09-38(A1)	0	Semiconfined	Theis Recovery	2.07	N/A
PZ9.5-4(A1)	46.7	Semiconfined	Theis	4.16	3.9 x 10 <sup>-3</sup>
PZ9.5-6(A2)	36.7	Semiconfined	Theis	3.65	1.5 x 10 <sup>-3</sup>
PT9-2(A1)	223	Semiconfined	Theis	2.58	1.7 x 10 <sup>-3</sup>
		A	2 Aquifer Test	· · · · · · · · · · · · · · · · · · ·	
W09-41(A2)	0	Semiconfined (Pumping data)	Theis Recovery Theis Partial Penetration	0.29 0.28	N/A N/A
PZ9.5-7(AQ)	20	Semiconfined	Theis Hantush Leaky (No Storage)	0.067 0.038	3.7 x 10 <sup>-3</sup> 2.8 x 10 <sup>-3</sup>
PZ9.5-1 (AQ)	53.7	Semiconfined	Theis Hantush Leaky (No Storage)	0.097 0.063	2.9 x 10 <sup>-4</sup> 2.5 x 10 <sup>-4</sup>
PT9-3(A2)	208	Semiconfined	Theis Hantush Leaky (No Storage)	1.60 0.78	1.5 x 10 <sup>-3</sup> 1.6 x 10 <sup>-3</sup>

### TABLE F4.4-3 A1/A2 AQUIFER ZONE HEAD DIFFERENTIALS PUMP TEST 5, SITE 9 MOFFETT FIELD

Well Pairs	Vertical Head Difference (ft.)	Elevation of Head (ft, msl)	Notes
W9-38,(A1), W9-41(A2)	0.02	14.13,14.33	vertical flow from A2 to A1

C:123R3MOF4.WKMj

#### **Table F4.4-4 Groundwater Analytical Results** Pump Test 5(A1), Site 9 **Moffett Field**

WELL No. W09-38 (A1)

Date Sample Taken Time Samples Taken			5/09/91 Quarterly Sampling 2Q91		12/02/91 13:00		12/03/91 00:30 7.3 Hours*		12/03/91 09:00 15.8 Hours		12/03/91 16:45 23.6 Hours	
COMPOUND	CRQL <sup>b</sup>	UNITs										
1,1,1-Trichloroethane	10	ug/L°	J <sup>d</sup>	52	J	9						
1,1-Dichloroethane	10	ug/L	J	71	J	16						l
1,1-Dichloroethene	10	ug/L	J	74	J	22	J	110	J	100	J	150
1,2-Dichloroethene (Total)	10	ug/L	ł	460	J	9		680		630		770
Methylene Chloride	10	ug/L	J	44		57	J	170				
Trichloroethene	10	ug/L		4,700		390		9,100		8,000		9,900

NOTES: <sup>a</sup> Pump test began at 1711 on 12/02/91. <sup>b</sup> CRQL = Contract Required Quantitation Limit.

c ug/L = parts per billion (ppb).
d J = Estimated value.

#### **Table F4.4-5 Groundwater Analytical Results** Pump Test 5(A2), Site 9 **Moffett Field**

WELL No. W09-41 (A2)

Date Sample Taken Time Samples Taken			Qı Sa	/09/91 parterly ampling 2Q91		2/05/91 00:15 3 Hours*		2/05/91 08:30 5.1 Hours		2/05/91 16:20 .9 Hours		2/05/91 16:30 Field iplicate
COMPOUND	CRQL <sup>b</sup>	UNITs										
1,1,1-Trichloroethane	10	ug/L°			J	38	J	48	J	54	J	47
1,1-Dichloroethane	10	ug/L			J	54	J	63	J	65	J	63
1,1-Dichloroethene	10	ug/L	Jd	170	J	100	J	110	J	110	J	88
1,2-Dichloroethene (Total)	) 10	ug/L		880		300		360		330		340
2-Butanone	10	ug/L							J	77		
Methylene Chloride	10	ug/L	J	220	J	58	J	59	J	64	J	47
Trichloroethene	10	ug/L		13,000		3,600		4,600				4,800

NOTES: <sup>a</sup> Pump test began at 1625 on 12/04/91. <sup>b</sup> CRQL = Contract Required Quantitation Limit.

c ug/L = parts per billion (ppb).
d J = Estimated value.

### TABLE F4.5-1 DESCRIPTION OF WELLS AND PIEZOMETERS PUMP TEST 8, SITE 9 MOFFETT FIELD

Well Designation	Well	Screened Interval (ft)	Filter Pack Interval (ft)	Casing Diam.(in)	Distance F Pumping		Pump	_	Comments
Designation	Voi	Tricital (11)	The var (19	Diamin		A2 Pump		A2	
Pumping Well	W09-35(A1)	14 to 24	12 to 25	4	N/A	5	Y	Y	Monitor w/ transducer
Pumping Well	W09-20(A2)	30 to 45	28 to 46.5	4	5	N/A	Υ	Y	Monitor w/ transducer
Piezometer	PZ9.8-1(AQ)	31 to 32	30.8 to 32.5	2	13.5	12.2	Y	Y	Monitor w/ transducer
Piezometer	PZ9.8-2(A1)	14.8 to 24.8	13.7 to 25.3	2	12.5	12.2	Υ	Υ	Monitor w/ transducer
Piezometer	PZ9.8-3(A2)	35 to 45	34 to 45.6	2	14.6	12.2	Y	Υ	Monitor w/ transducer
Plezometer	PZ9.8-4(A1)	9.5 to 19.5	8 to 19.5	2	30.2	30.2	Y	Υ	Monitor w/ transducer
Piezometer	PZ9.8-5(A2)	30 to 45	28.5 to 45	2	30.2	30.2	Y	Υ	Monitor w/ transducer
Piezometer	PZ9.8-6(A1)	9.5 to 19.5	8.5 to 22	2	53.5	48.5	Y	Υ	Monitor w/ transducer
Piezometer	PZ9.8-7(A2)	30 to 40	29 to 40	2	53.5	48.5	<u>Y</u>	Υ	Monitor w/ transducer
Monitoring Well	W09-14(A2)	39 to 49	36.8 to 52	4	346	346	N	Y	Monitor w/ W.L. meter
Monitoring Well	W09-15(B2)	87.3 to 97.3	81.2 to 108	4	92	92	Y	Υ	Monitor w/ W.L. meter
Monitoring Well	W09-17(A2)	33 to 38	31 to 40	4	476	476	N	Y	Monitor w/ W.L. meter
Monitoring Well	W09-18(A1)	14 to 24	12 to 25	4	387	387	N	Υ	Monitor w/ W.L. meter
Monitoring Well	W09-21(A2)	41 to 46	39 to 48	4	243	243	N	Y	Monitor w/ W.L. meter
Monitoring Well	W09-23(A1)	8 to 18	6 to 20	4	325	321	N	N	Monitor w/ W.L. meter
Monitoring Well	W09-28(A2)	38 to 48	36 to 50	4	337	334	N	Υ	Monitor w/ W.L. meter
Monitoring Well	W09-33(A2)	34 to 49	32 to 51.5	4	305	305	N	Υ	Monitor w/ W.L. meter
Monitoring Well	W09-36(A2)	33 to 43	28 to 44	4	336	336	N	Υ	Monitor w/ W.L. meter
Monitoring Well	W09-37(A1)	10 to 20	8 to 21.5	4	582	582	N	N	Monitor w/ W.L. meter

Notes:

N/A: Not Applicable PZ: Piezometer

W.L.: Water Levoi meter

Table F4.5-2

Aquifer Classification and Pump Test Analytical Results
Pump Test 8, Site 9
Moffett Field

Well Number	Distance From Pump	Aquifer Classification	·		Storativity
		A	1 Aquifer Test		
W09-35(A1)	0	Semiconfined (Unconfined, pumping data)	Theis Recovery Cooper-Jacob	0.54 0.15	N/A 4.3 x 10 <sup>-3</sup>
PZ9.8-2(A1)	12.5	Semiconfined	Theis Hantush Leaky (No Storage)	0.11 0.074	6.2 x 10 <sup>-3</sup> 5.1 x 10 <sup>-3</sup>
PZ9.8-4(A1)	30.2	Semiconfined	Theis Hantush Leaky (No Storage)	0.82 0.74	4.0 x 10 <sup>-3</sup> 4.0 x 10 <sup>-3</sup>
PZ9.8-6(A1)	53.5	Semiconfined	Theis Hantush Leaky (No Storage)	1.1 0.94	4.7 x 10 <sup>-4</sup> 6.4 x 10 <sup>-4</sup>
		Α	2 Aquifer Test		
W09-20(A2)	0	Semiconfined	Theis Recovery	0.47	N/A
PZ9.8-3(A2)	12.2	Semiconfined	Hantush Leaky (No Storage)	0.33	0.066
PZ9.8-5 (A2)	30.2	Semiconfined	Theis Hantush Leaky (No Storage)	0.23 0.28	5.1 x 10 <sup>-4</sup> 5.1 x 10 <sup>-4</sup>
PZ9.8-7(A2)	48.5	Semiconfined	Theis Hantush Leaky (No Storage)	0.21 0.21	2.8 x 10 <sup>-4</sup> 2.8 x 10 <sup>-4</sup>
PZ9.8-1(AQ)	12.2	Semiconfined	Hantush Leaky (No Storage)	0.11	2.3 x 10 <sup>-3</sup>

### TABLE F4.5-3 A1/A2 AQUIFER ZONE HEAD DIFFERENTIALS PUMP TEST 8, SITE 9 MOFFETT FIELD

Well Pairs	Vertical Head Difference (ft.)	Elevation of Head (ft, msl)	Notes
W9-35(A1), W9-20(A2)	0.08	10.11, 10.19	vertical flow from A2 to A1

CA123R3AMOP3.WXSAj

#### **Table F4.5-4 Groundwater Analytical Results** Pump Test 8(A1), Site 9 **Moffett Field**

WELL No. W09-35 (A1)

Date Sample Taken Time Samples Taken	Qua San	13/91 arterly npling Q91	11/14/91 14:00 5 Hours*			
COMPOUND	CRQL <sup>b</sup>	UNITs				
1,1,1-Trichloroethane	10	ug/L°	J <sup>d</sup>	36	J	40
1,1-Dichloroethane	10	ug/L	J	53	J	39
1,1-Dichloroethene	10	ug/L	J	110	J	57
1,2-Dichloroethene (Total)	) 10	ug/L		430		460
Methylene Chloride	10	ug/L	!		J	140
Tetrachloroethene	10	ug/L	] J	120	J	56
Trichloroethene	10	ug/L	J	120		3,100

NOTES: <sup>a</sup> Pump test began at 0902 on 11/14/91. <sup>b</sup> CRQL = Contract Required Quantitation Limit.

c ug/L = parts per billion (ppb).
d J = Estimated value.

#### **Table F4.5-5 Groundwater Analytical Results** Pump Test 8 (Á2), Site 9 Moffett Field

WELL No. W09-20 (A2)

Date Sample Taken Time Samples Taken			Quart	5/13/91 erly Sampling 2Q91		1/19/91 21:20 3 Hours		1/20/91 05:30 Hours		1/20/91 13:00 5 Hours
COMPOUND	CRQL <sup>b</sup>	UNITs								
1,1,1-Trichloroethane	10	ug/L°					J	120	J	130
1,1-Dichloroethene	10	ug/L	J <sup>d</sup>	260	J	190	J	200	J	190
1,2-Dichloroethene (Total)	10	ug/L	J	420	J	370	J	360	J	380
Methylene Chloride	10	ug/L	J	170	J	380	J	280	J	370
Tetrachloroethene	10	ug/L	J	480	J	280	J	260	J	260
Trichloroethene	10	ug/L		18,000		12,000		13,000		14,000

NOTES: a Pump test began at 1332 on 11/19/91.

b CRQL = Contract Required Quantitation Limit.
c ug/L = parts per billion (ppb).
d J = Estimated value.

#### Table F5-1

### Summary of Hydraulic Parameters Pump Test 7, Site 8 Moffett Field

Pump Test	Observation Well Piezometer	Transmissivity (ft²/min)	Hydraulic Conductivity (ft/min)	Storativity	Estimated Average Thickness (ft)	Aquifer Type	Analysis Method
7	PZ8.7-1(A1) <sup>a</sup>	1.42	0.095	6.2 x 10 <sup>-2</sup>	15	Semiconfined	Hantush (1960)
7	PZ8.7-2(A1) <sup>a</sup>	2.43	0.16	9.2 x 10⁴	15	Semiconfined	Hantush and Jacob (1955)
7	W08-10(A1)	1.24	0.83	3.8 x 10 <sup>-5</sup>	15	Semiconfined	Hantush (1960)
7	W08-12(A1)	1.00	0.67	1.1 x 10⁴	15	Semiconfined	Hantush (1960)
Geometric Mean		1.44	0.10	2.2 x 10⁴			

<sup>&</sup>lt;sup>a</sup>Where two methods were used for data evaluation (i.e., Cooper-Jacob-Modified versus Hantush Leaky Type Curve), the results from the method best fitting the data was presented.

NA - Not applicable.

References:

Hantush, M. S. and C. E. Jacob, 1955, Non-Steady Radial Flow in an Infinite.

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Geophys. Res., Vol. 65, No. 11, pp. 3713-3725.

Table F5-2

#### Summary of Hydraulic Parameters Pump Tests 1, 3, 5, and 8, Site 9 Moffett Field

(Page 1 of 2)

Pump Test	Observation Well Piezometer	Aquifer Zone	Transmissivity (ft <sup>2</sup> /min)	Hydraulic Conductivity (ft/min)	Storativity	Estimated Average Thickness (ft)	Aquifer Type	Analysis Method
1	PZ9.1-2(A2)	A2	4.36	0.36	0.0034	12	Semiconfined	Hantush & Jacob (1955)
1	Pz9.1-3(A2)	A2	9.78	0.82	0.0016	12	Semiconfined	Hantush & Jacob (1955)
1	W09-13(A2)	<b>A</b> 2	16.76	1.40	0.0002	12	Semiconfined	Hantush & Jacob (1955)
1	W09-25(A2)	A2	36.81	3.07	0.003	12	Semiconfined	NA
Geometric Mean	NA	A2	12.74	1.06	0.0013	12	Semiconfined	NA
3	PZ9-3-3(A2)	A2	2.31	0.39	0.0040	6	Semiconfined	Hantush & Jacob (1955)
3	PZ9.3-5(A2)	<b>A</b> 2	2.21	0.37	0.0020	6	Semiconfined	Hantush & Jacob (1955)
3	W29-08(A2)	A2	3.56	0.59	0.0100	6	Semiconfined	Hantush & Jacob (1955)
Geometric Mean	NA	<b>A</b> 2	2.63	0.44	0.0040	6	Semiconfined	NA
5	PT9.2(A1)	A1	25.32	1.95	0.011	13	Semiconfined	Hantush & Jacob (1955)
5	PZ9.5-4(A1)	<b>A</b> 1	29.55	2.27	0.029	13	Semiconfined	Hantush & Jacob (1955)
5	PZ9.5-6(A1)	A1	29.39	2.26	0.010	13	Semiconfined	Hantush & Jacob (1955)
Geometric Mean	NA	<b>A</b> 1	28.02	2.16	0.015	13	Semiconfined	NA
5	PZ9.5-1(AQ)	A2	0.48	0.08	0.0019	6	Semiconfined	Hantush & Jacob (1955)
5	PT9-3(A2)	<b>A</b> 2	5.83	0.97	0.0107	6	Semiconfined	Hantush & Jacob (1955)

Table F5-2

(Page 2 of 2)

Pump Test	Observation Well Piezometer	Aquifer Zone	Transmissivity (ft <sup>2</sup> /min)	Hydraulic Conductivity (ft/min)	Storativity	Estimated Average Thickness (ft)	Aquifer Type	Analysis Method
5	PZ9.5-7(A2)	A2	0.29	0.05	0.0214	6	Semiconfined	Hantush & Jacob (1955)
Geometric Mean	NA	A2	0.93	0.16	0.0076	6	Semiconfined	NA
8	PZ9.8-2(A1)	<b>A</b> 1	0.54	0.07	0.0380	8	Semiconfined	Hantush & Jacob (1955)
8	PZ9.8-4(A1)	<b>A</b> 1	5.87	0.73	0.0299	8	Semiconfined	Hantush & Jacob (1955)
8	PZ9.8-6(A1)	<b>A</b> 1	2.26	0.28	0.0201	8	Semiconfined	Hantush & Jacob (1955)
Geometric Mean	NA	A1	1.93	0.24	0.0284	8	Semiconfined	NA
8	PZ9.8-1(A2)	A2	2.64	0.26	0.0499	10	Semiconfined	Hantush & Jacob (1955)
8	PZ9.8-3(A1)	A2	2.44	0.24	0.4861	10	Semiconfined	Hantush & Jacob (1955)
8	PZ9.8-5(A2)	A2	1.95	0.20	0.0039	10	Semiconfined	Hantush & Jacob (1955)
8	PZ9.8-7(A2)	A2	1.56	0.16	0.0022	10	Semiconfined	Hantush & Jacob (1955)
Geometric Mean	NA	A2	2.10	0.21	0.0304	10	Semiconfined	Hantush & Jacob (1955)
Geometric Mean	Site 9	A1	7.35	0.72	0.0206	10.20	Semiconfined	NA
Geometric Mean	Site 9	A2	2.84	0.35	0.0024	8.11	Semiconfined	NA

NA - Not applicable

References:

Hantush, M. S. and C. E. Jacob, 1955, Non-Steady Radial Flow in an Infinite Leaky Aquifer, Am. Geophys. Union Trans.,

Vol. 36, pp. 95-100.

Hantush, M. S., 1960, Modification of the Theory of Leaky Aquifers, Joun. of Geophys. Res., Vol. 65, No. 11

рр 3713-3725.

#### Table F5-3

#### Summary of Hydraulic Parameters A1/A2 Aquitard, Site 9 Moffett Field

Pump Test	Piezometer	Transmissivity (ft²/min)	Hydraulic Conductivity (ft/min)	Storativity	Assumed Thickness (ft)	Analysis Method
1	PZ9.1-1(AQ)	5.0 x 10 <sup>-4</sup>	9.07 x 10 <sup>-5</sup>	1.0 x 10 <sup>-5</sup>	5.5	Cooper, et al. (1967)
1	PZ9.1-1(AQ)	7.8 x 10⁴	1.4 x 10 <sup>-4</sup>	6.1 x 10⁴	5.5	Neuman and Whitherspoon (1969)
3	PZ9.3-1(AQ)	3.6 x 10 <sup>-3</sup>	2.25 x 10 <sup>4</sup>	5.0 x 10 <sup>-5</sup>	17.0	Cooper, et al. (1967)
3	PZ9.3-1(AQ)	3.1 x 10 <sup>-3</sup>	1.84 x 10⁴	2.0 x 10 <sup>-3</sup>	17.0	Neuman and Whitherspoon (1969)
8ª	PZ9.8-1(AQ)	1.1 x 10 <sup>-2</sup>	1.00 x 10 <sup>-3</sup>	3.8 x 10 <sup>-6</sup>	11.0	Cooper, et al. (1967) <sub>b</sub>
Geometric Mean	NA	2.12 x 10 <sup>-3</sup>	2.19 x 10 <sup>-4</sup>	4.05 x 10⁴	9.7	NA

NA - Not applicable

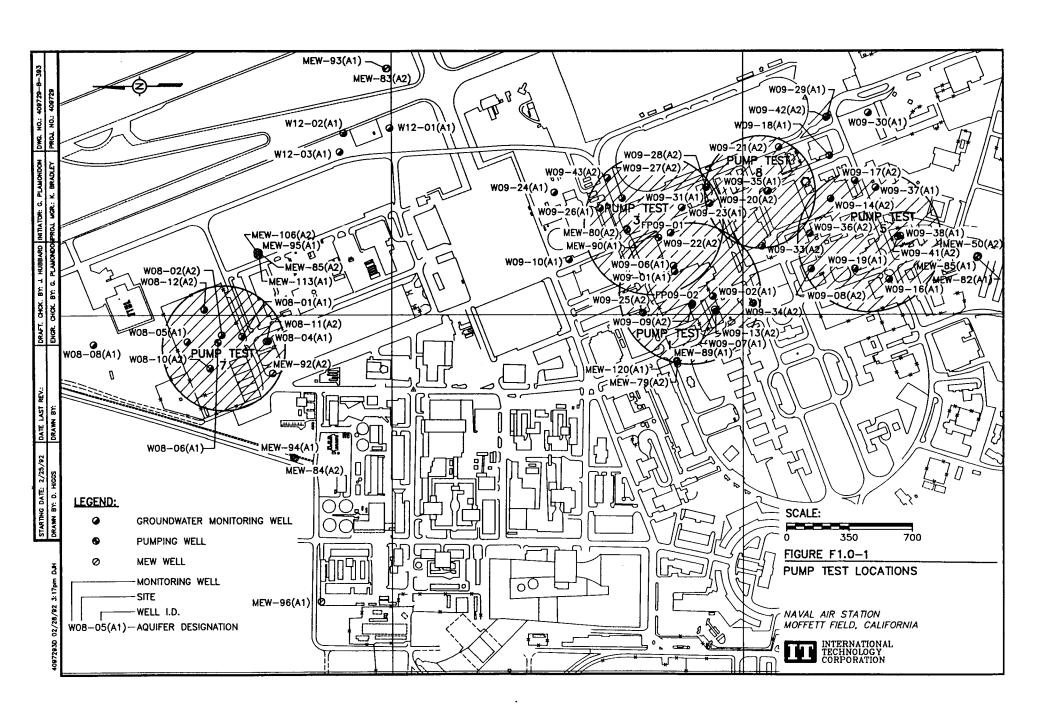
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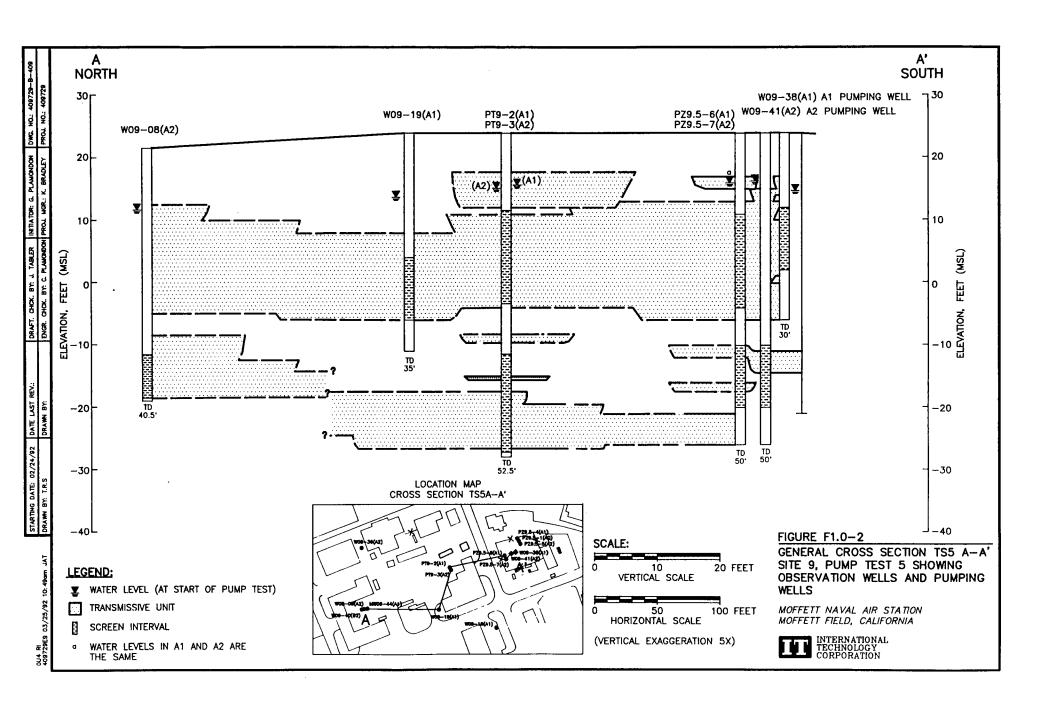
Cooper, H. H., Jr., Bredehoeft, and I. S. Papadopulos, 1967, Response of a Finite Diameter Well to an Instantaneous Change in Water, Water Resources Research, Vol. 3, No. 1, pp. 263-269.

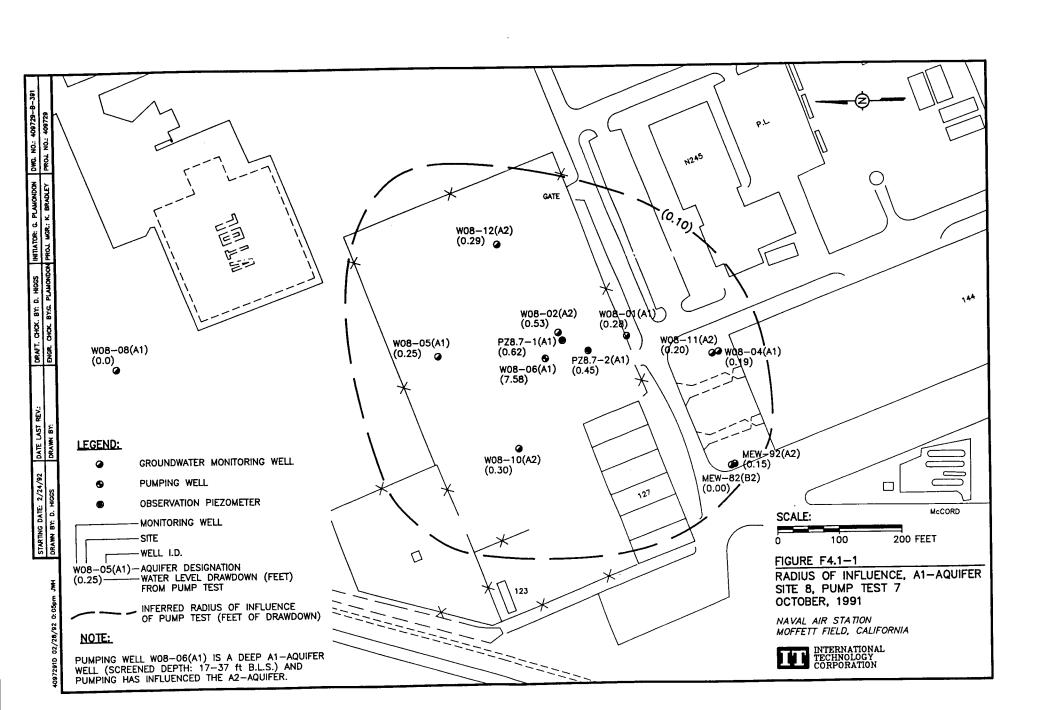
Neuman, S. P., and P. A. Whitherspoon, 1969, Application of Curernt Theories of Flow in Leaky Aquifers, Water Resources Research,

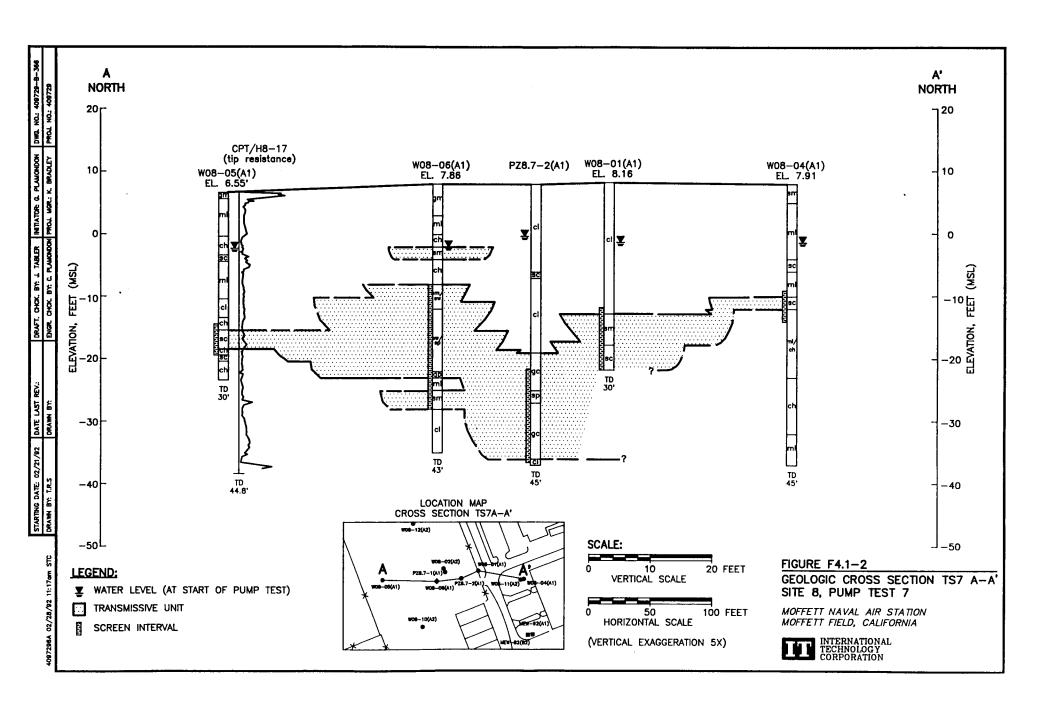
Vol. 5, No. 4, pp. 817-829.

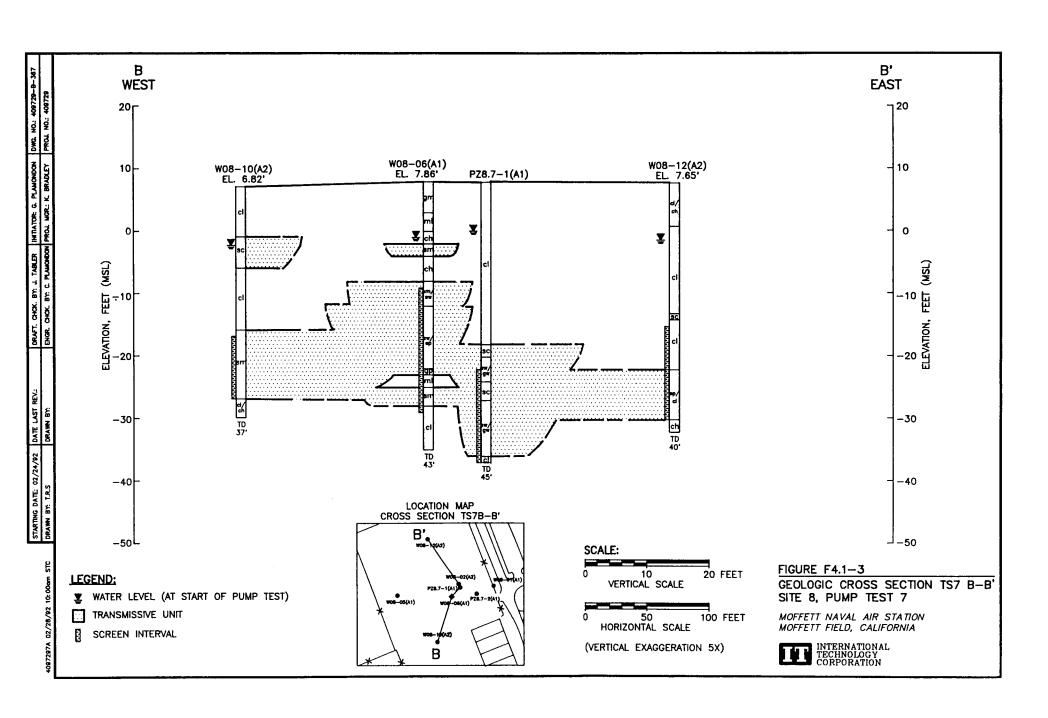
<sup>\*</sup>Not used to calculate geometric mean. Piezometer hydraulically connected to the pumping well.

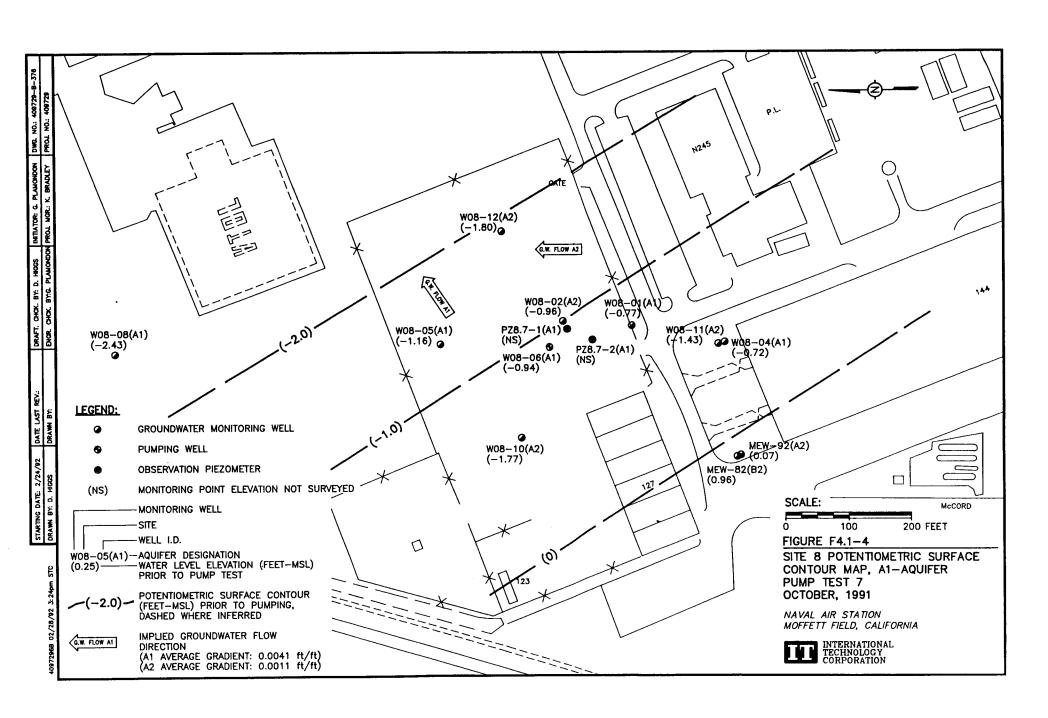


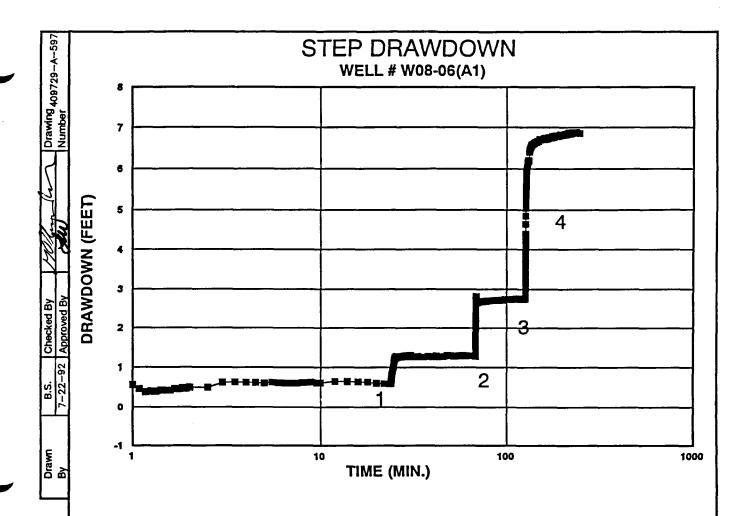












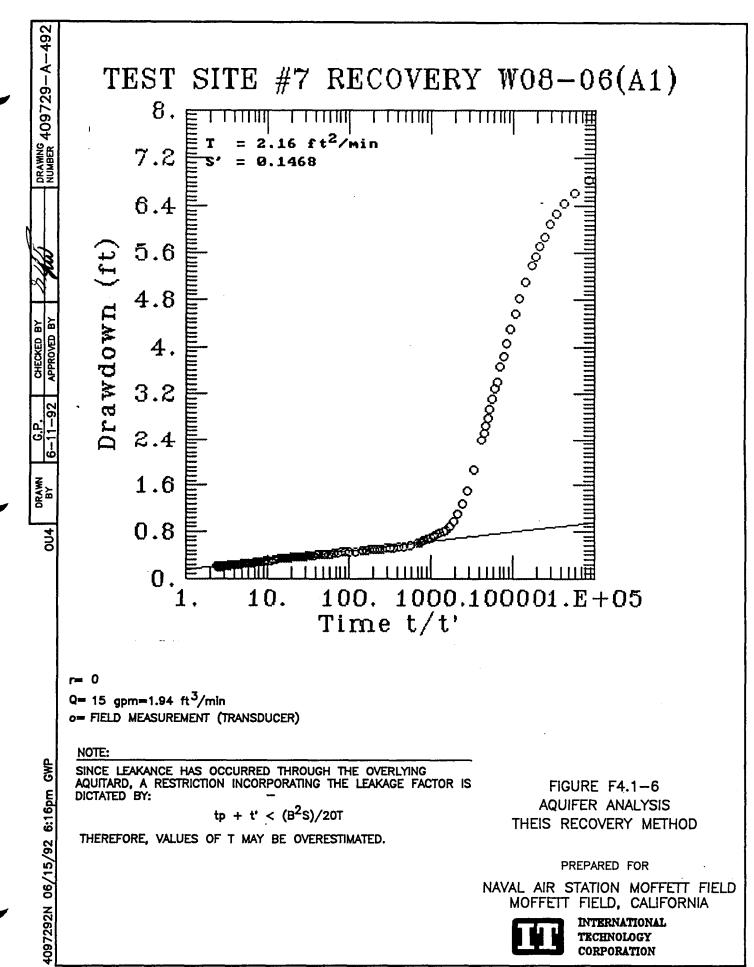
STEP#	DISCHARGE RATE	CUMLATIVE DRAWDOWN	SPECIFIC CAPACITY
	Q (GPM)	s (FEET)	Q/s (gpm/ft)
1	2.1	0.61	3.621
2	4.5	1.30	3.516
3	8.5	2.75	3.137
4	16.5	6.87	2.412

FIGURE F4.1-5

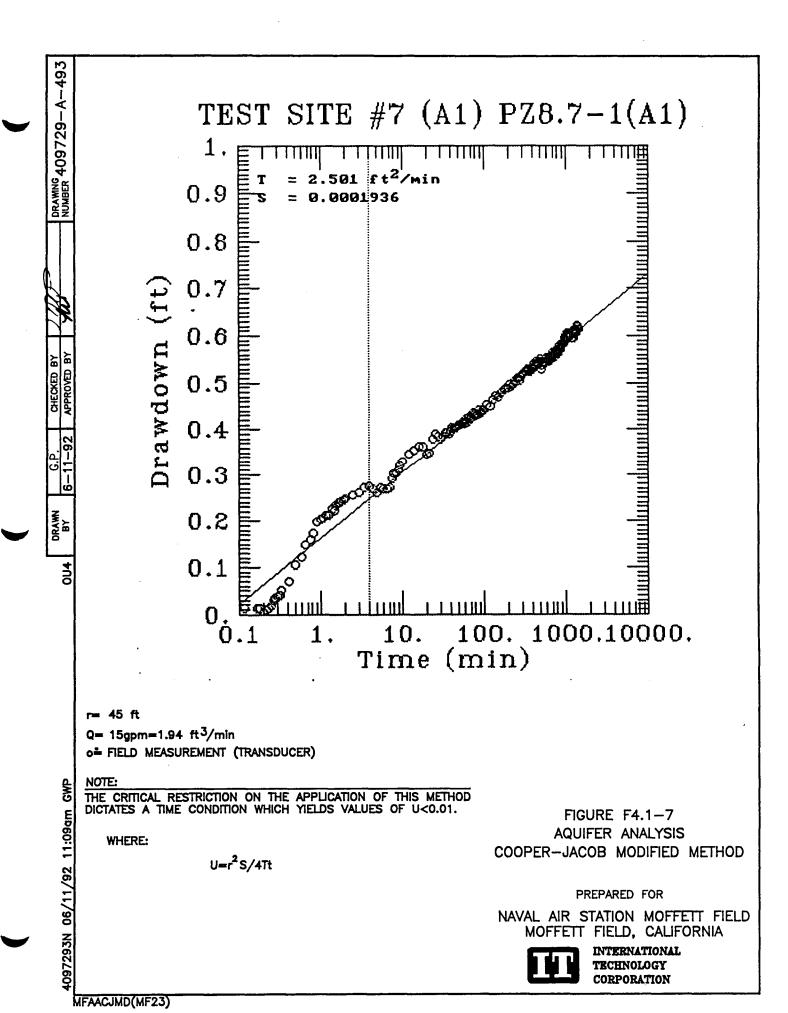
Pump Test 7 Step-Drawdown Test

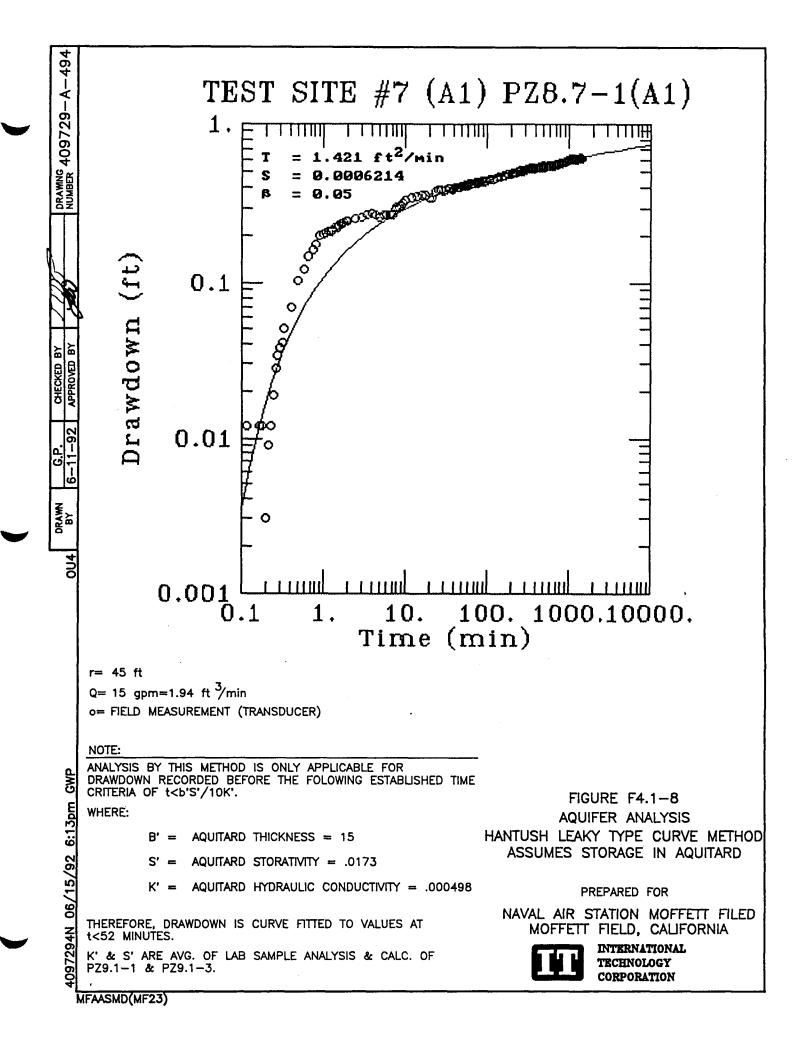
NAVAL AIR STATION MOFFET FIELD, CALIFORNIA

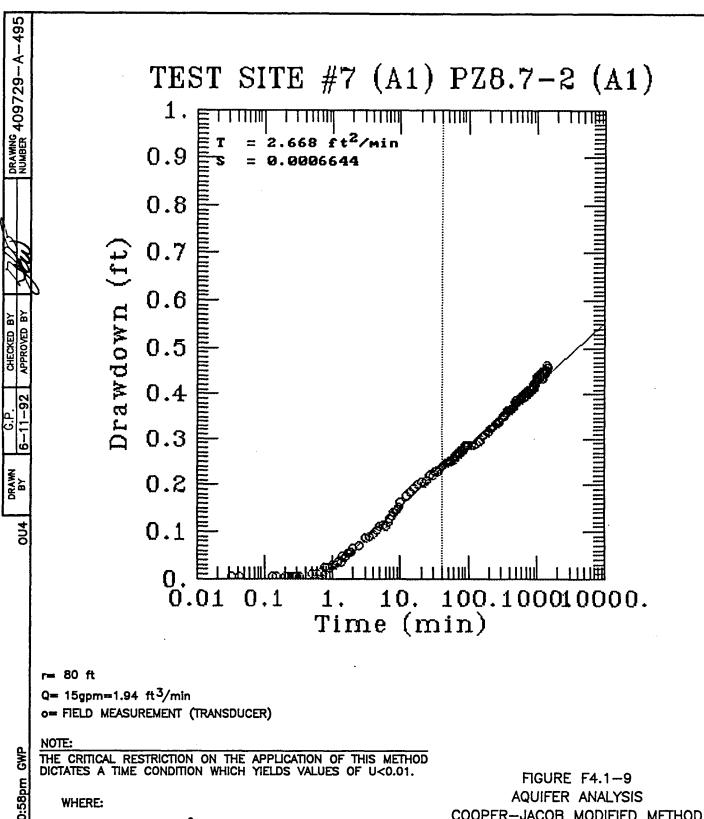




MFAATRMD(MF23)







 $U=r^2S/4Tt$ 

THEREFORE, APPLICABLE VALUES ARE AT t> 40 min.

COOPER-JACOB MODIFIED METHOD

PREPARED FOR

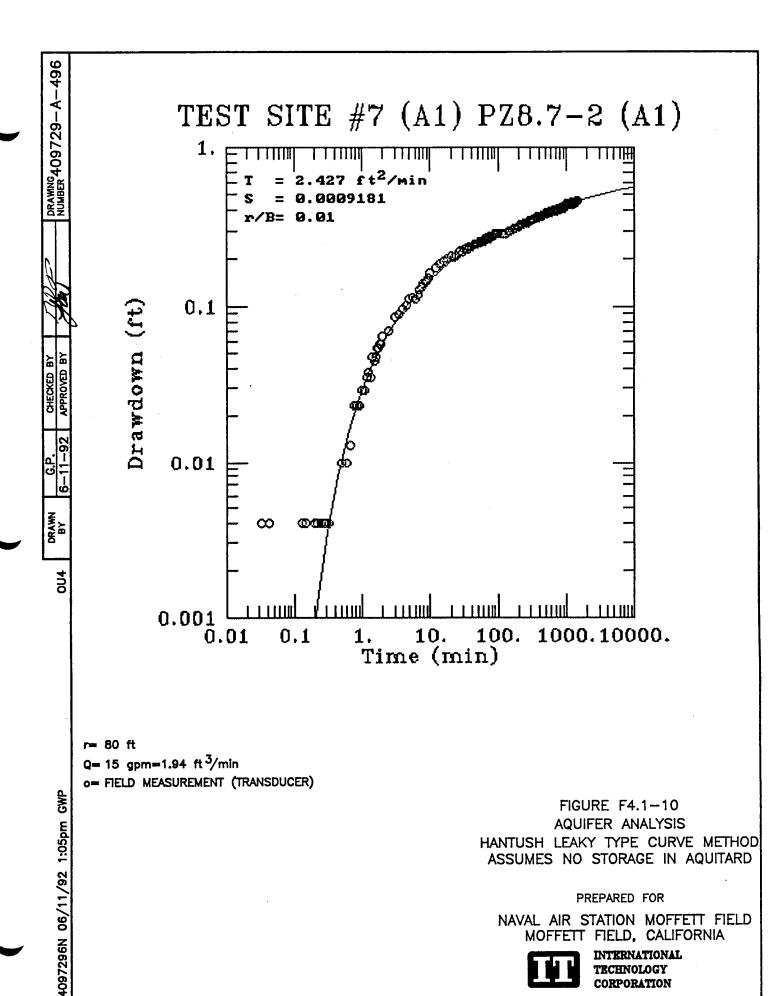
NAVAL AIR STATION MOFFETT FIELD MOFFETT FIELD, CALIFORNIA



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H097295N 06/11/92

MFAACJMD(MF23)



TEST SITE #7 W08-10(A2) 409729-= 3.8007E-05= 1.0090.1 0.01 0.001 0.01 0.1 10. 100. 1000.10000. Time (min) r= 153 ft Q= 15 gpm=1.94 ft  $\frac{3}{min}$ o= FIELD MEASUREMENT (TRANSDUCER) NOTE: ANALYSIS BY THIS METHOD IS ONLY APPLICABLE FOR DRAWDOWN RECORDED BEFORE THE FOLOWING ESTABLISHED TIME FIGURE F4.1-11 CRITERIA OF t<b'S'/10K'. AQUIFER ANALYSIS

WHERE:

AQUITARD THICKNESS = 10

AQUITARD STORATIVITY = .0173

AQUITARD HYDRAULIC CONDUCTIVITY = .000498

HANTUSH LEAKY TYPE CURVE METHOD ASSUMES STORAGE IN AQUITARD

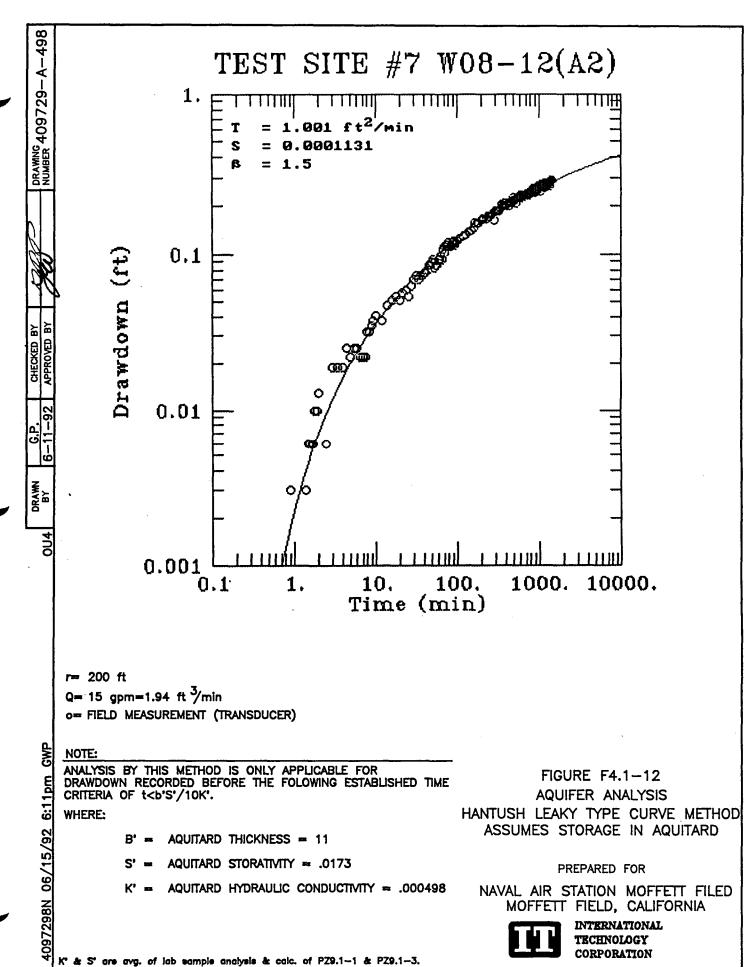
PREPARED FOR

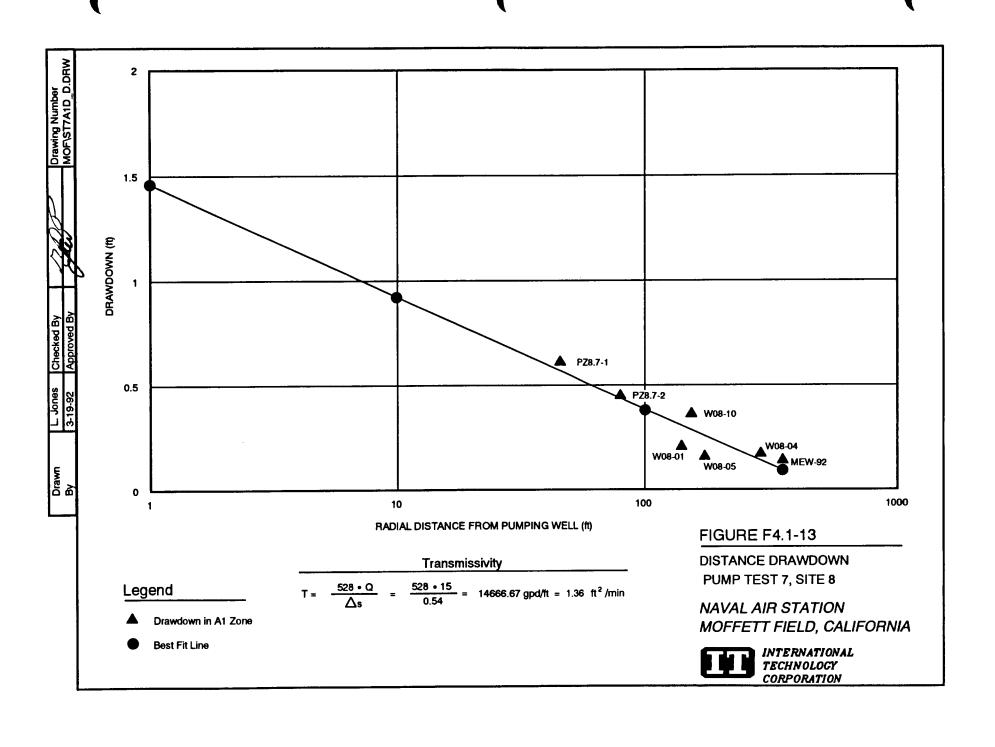
NAVAL AIR STATION MOFFETT FILED MOFFETT FIELD, CALIFORNIA

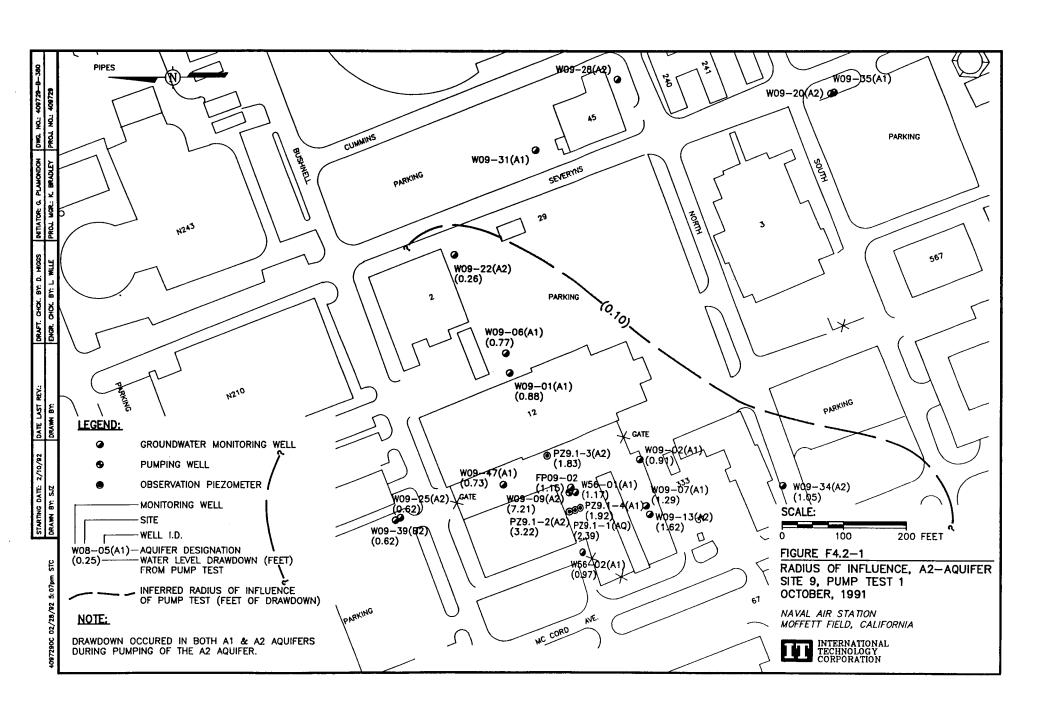


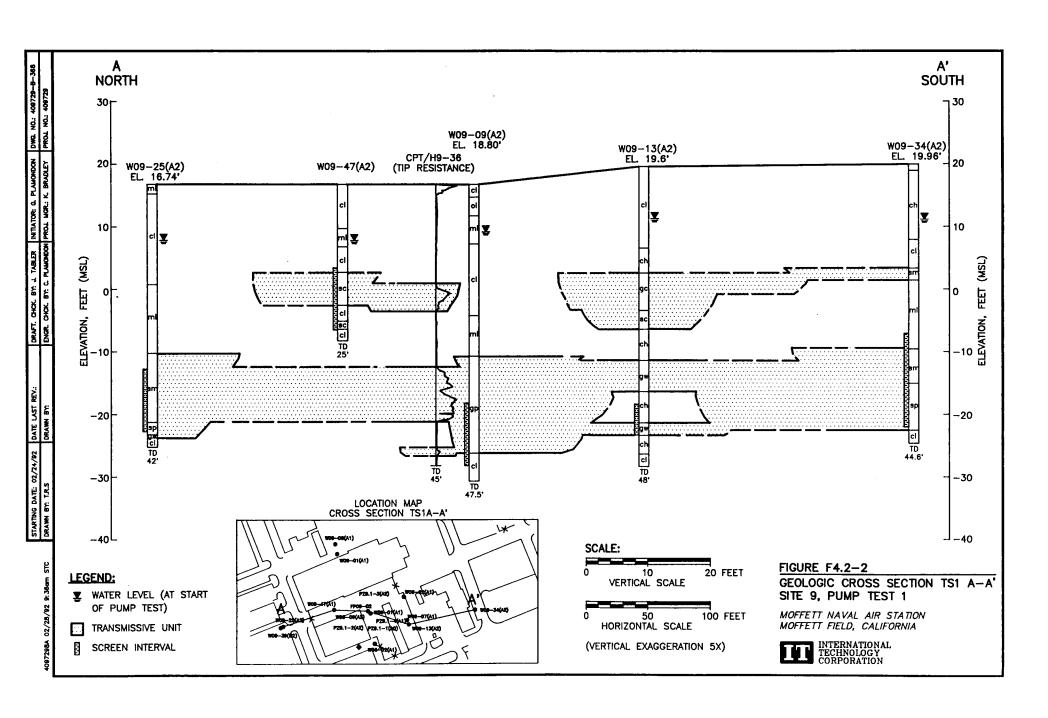
INTERNATIONAL TECHNOLOGY CORPORATION

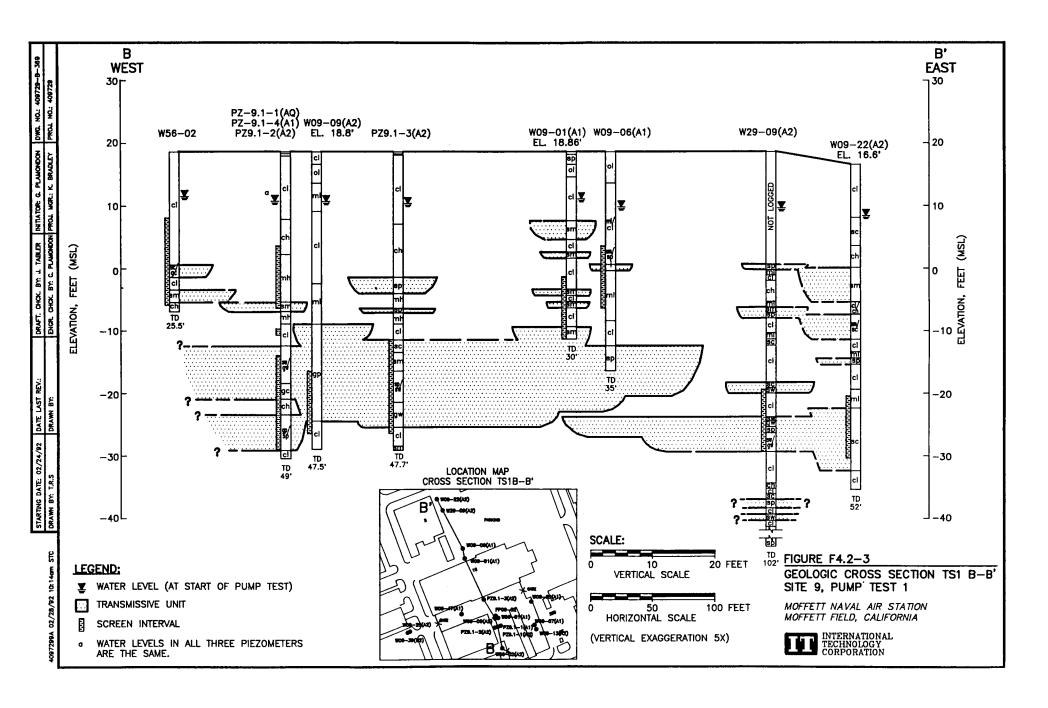
THEREFORE, DRAWDOWN IS CURVE FITTED TO VALUES AT t< 35 MINUTES

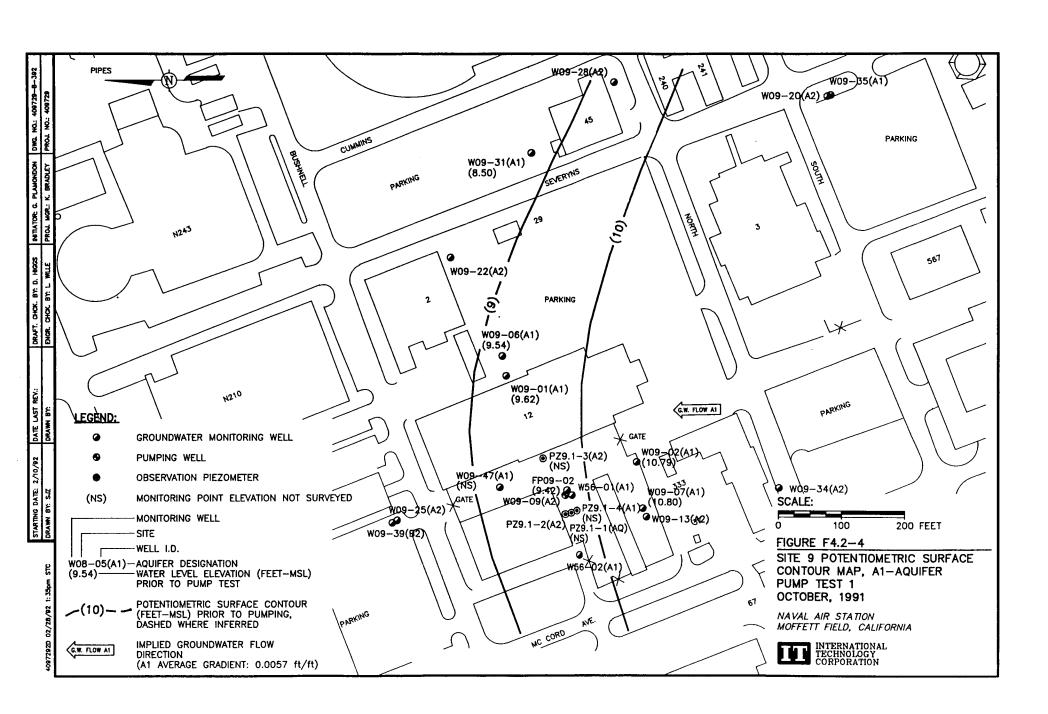


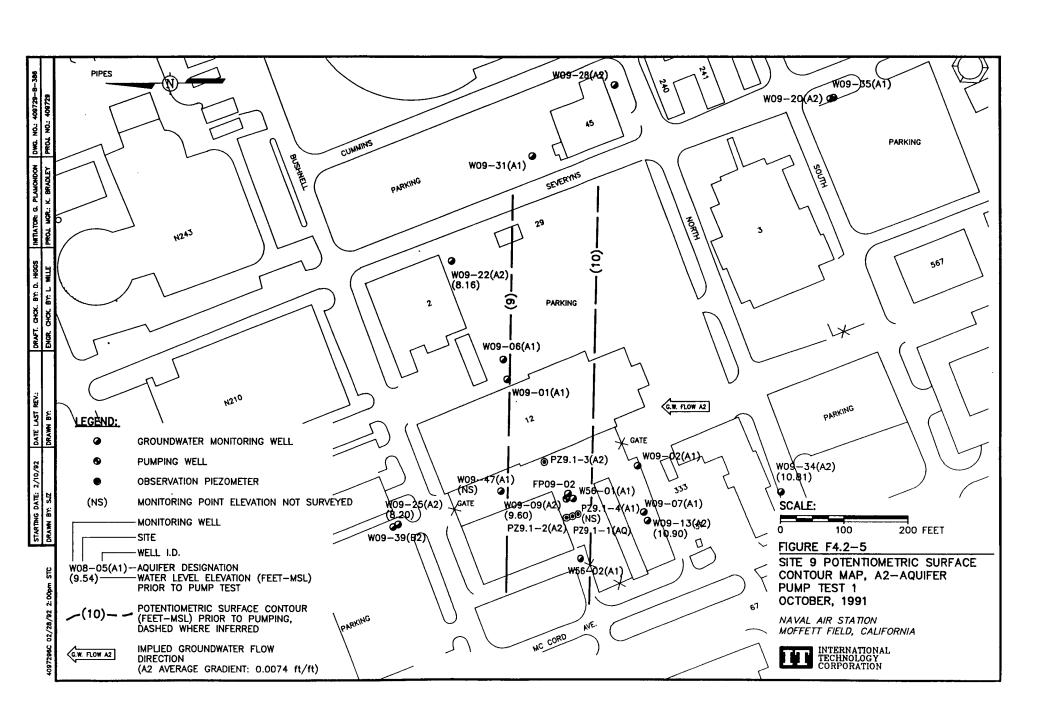


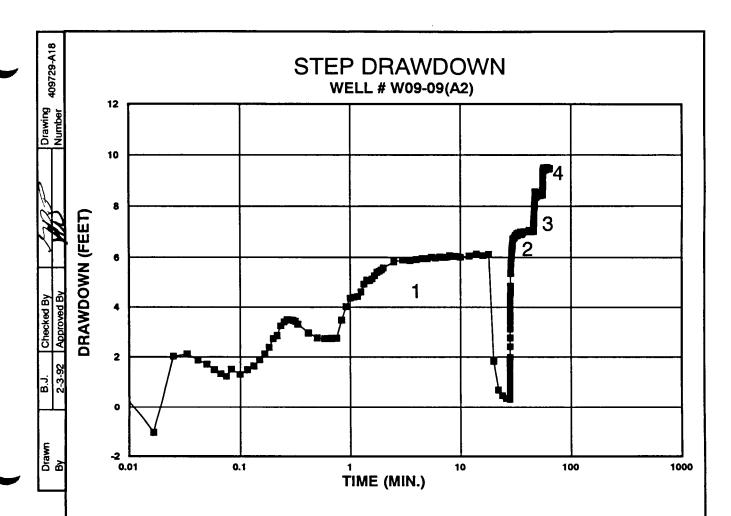












STEP#	DISCHARGE RATE Q (GPM)	CUMULATIVE DRAWDOWN s (feet)	SPECIFIC CAPACITY Q/s (gpm/ft)
1	36	6.09	5.911
2	41	7.00	5.857
3	50	8.50	5.882
4	58	9.45	6.138

Note:

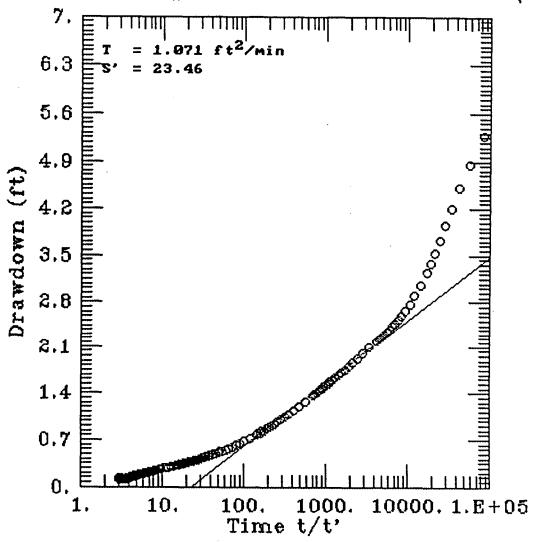
The initial step-drawdown test included discharge rates ranging from 1.8 gpm to 28.8 gpm and is not included in the graph.

FIGURE F4.2-6

Pump Test 1 Step-Drawdown Test

NAVAL AIR STATION MOFFETT FIELD, CALIFORNIA





r= 0

Q= 42 gpm=5.61 ft<sup>3</sup>/min

o= FIELD MEASUREMENT (TRANSDUCER)

NOTE:

SINCE LEAKANCE HAS OCCURRED THROUGH THE OVERLYING AQUITARD, A RESTRICTION INCORPORATING THE LEAKAGE FACTOR IS DICTATED BY:

 $tp + t' < (B^2S)/20T$ 

THEREFORE, VALUES OF T MAY BE OVERESTIMATED.

FIGURE F4.2-7
AQUIFER ANALYSIS
THEIS RECOVERY METHOD

PREPARED FOR

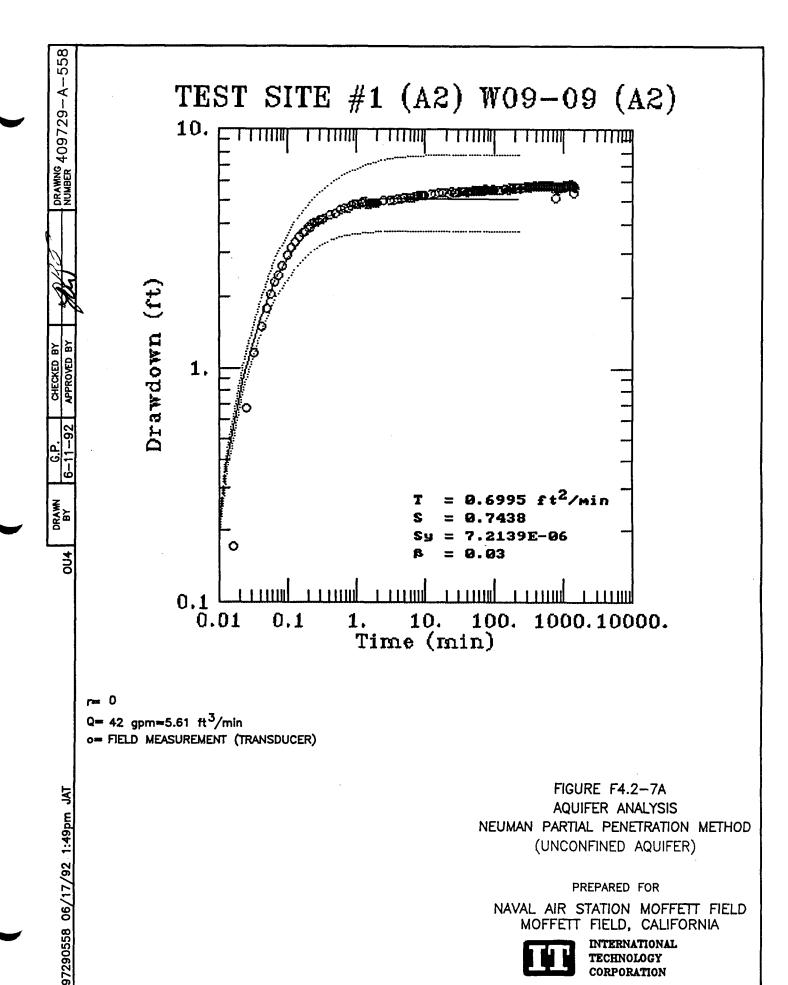
NAVAL AIR STATION MOFFETT FIELD MOFFETT FIELD, CALIFORNIA



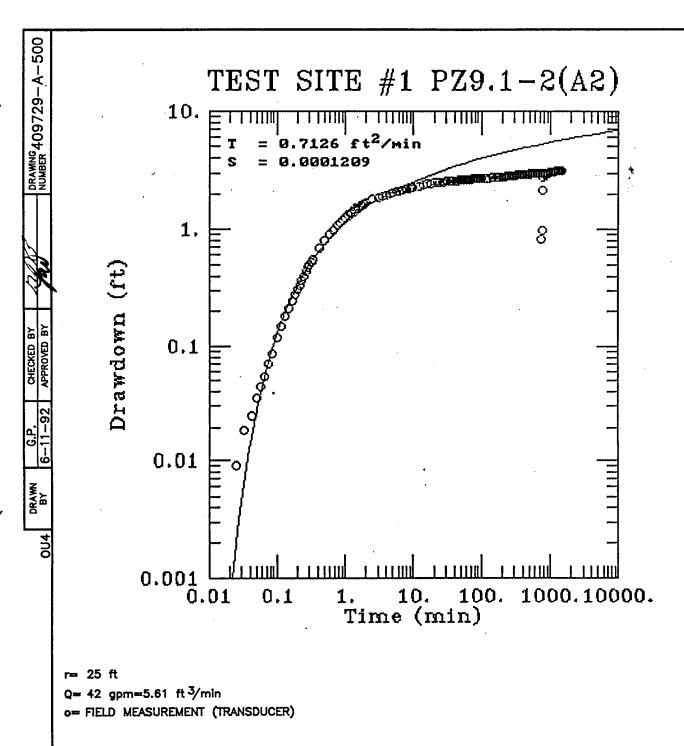
INTERNATIONAL TECHNOLOGY CORPORATION

H097299N 06/15/92 10:09am GWP

MFAATRMD(MF23)



MFAATRMD(MF23)



NOTE:

BASED ON LITHOLOGY AND LEAKY TIME-DRAWDOWN RESPONSE, THIS METHOD IS CONSIDERED VALID FOR DRAWDOWN BEFORE STORAGE IS RELEASED FROM THE AQUITARD.

FIGURE F4.2-8
AQUIFER ANALYSIS
THEIS TYPE CURVE METHOD

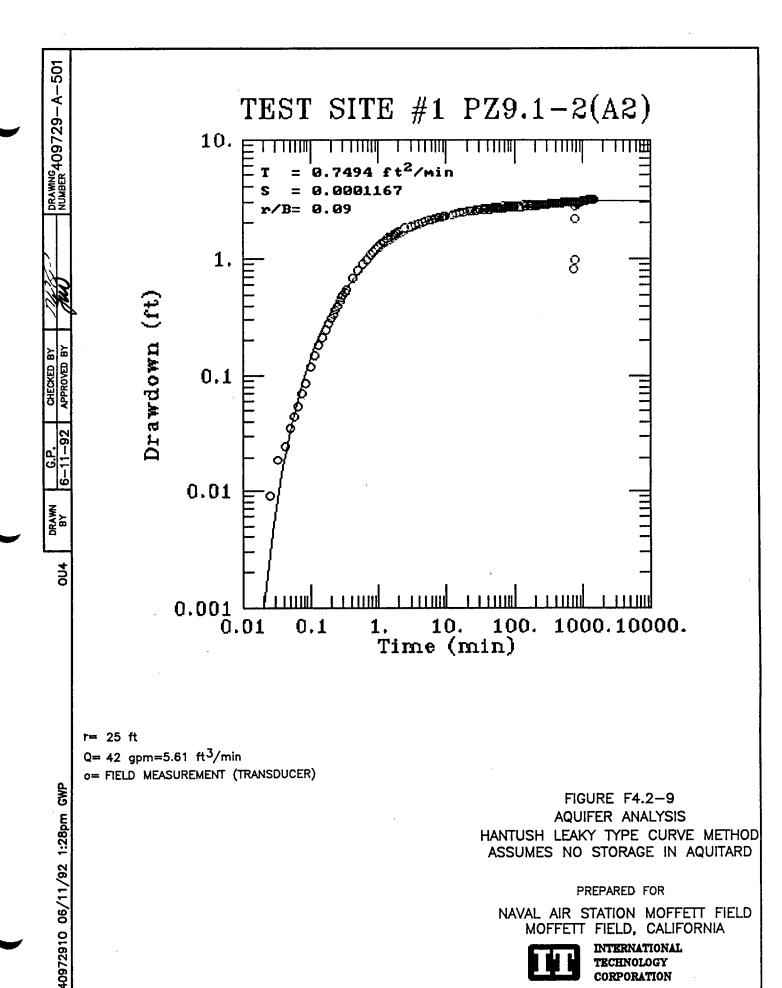
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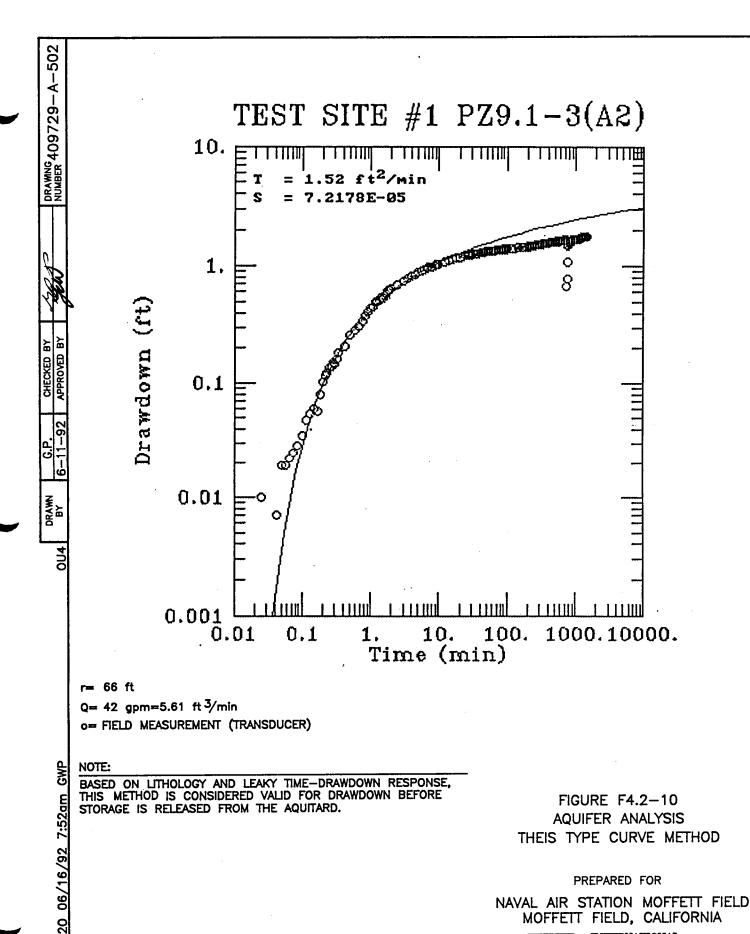
NAVAL AIR STATION MOFFETT FIELD MOFFETT FIELD, CALIFORNIA



INTERNATIONAL TECHNOLOGY CORPORATION

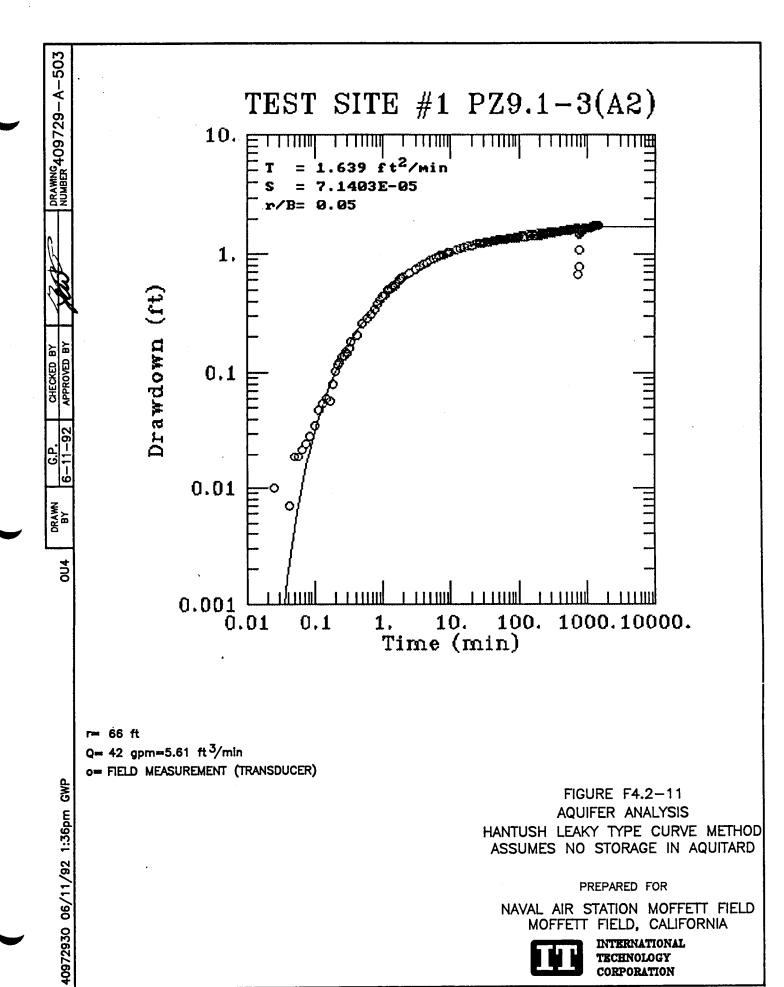
772900 06/16/92 7.54cm CW

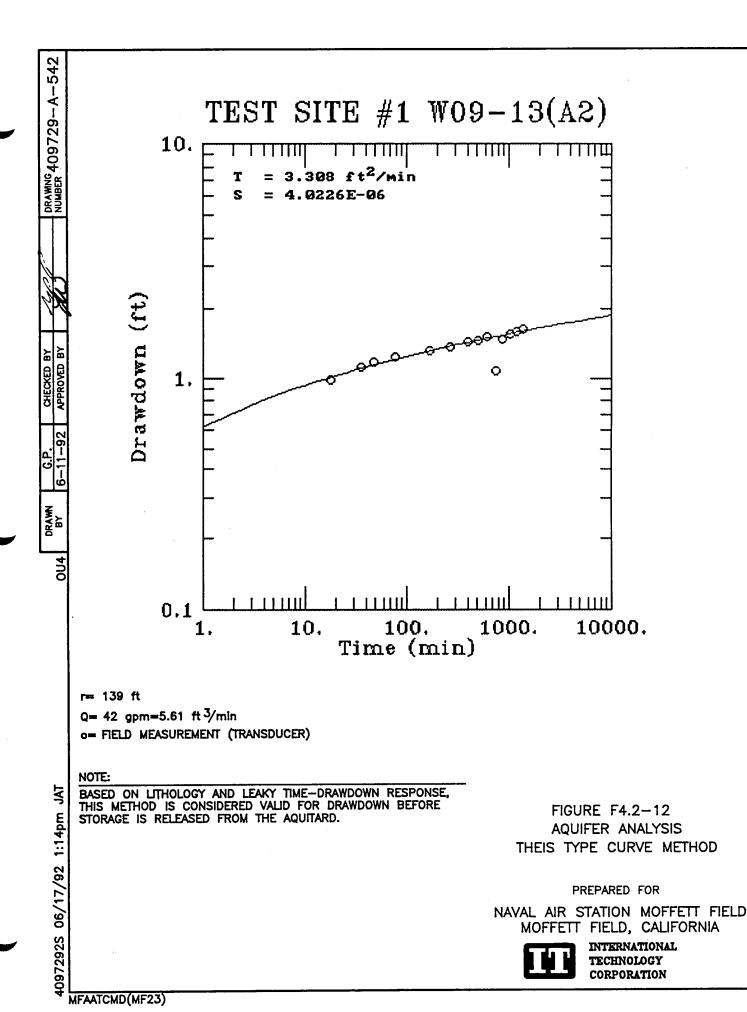


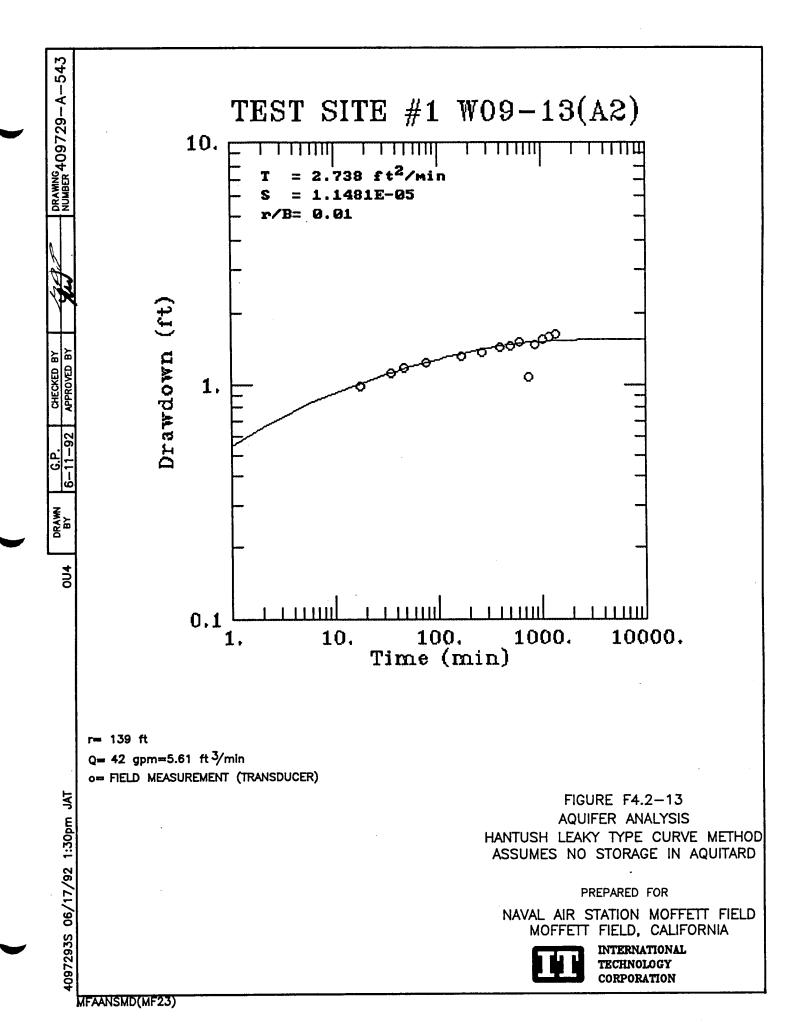


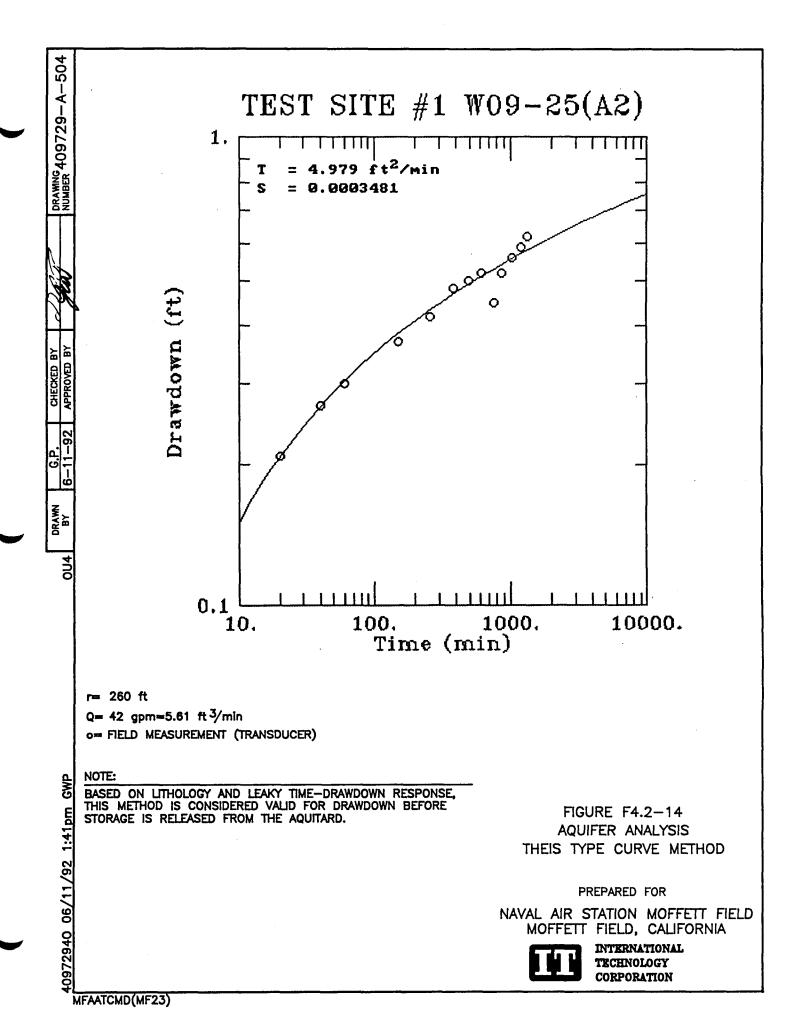
INTERNATIONAL TECHNOLOGY CORPORATION

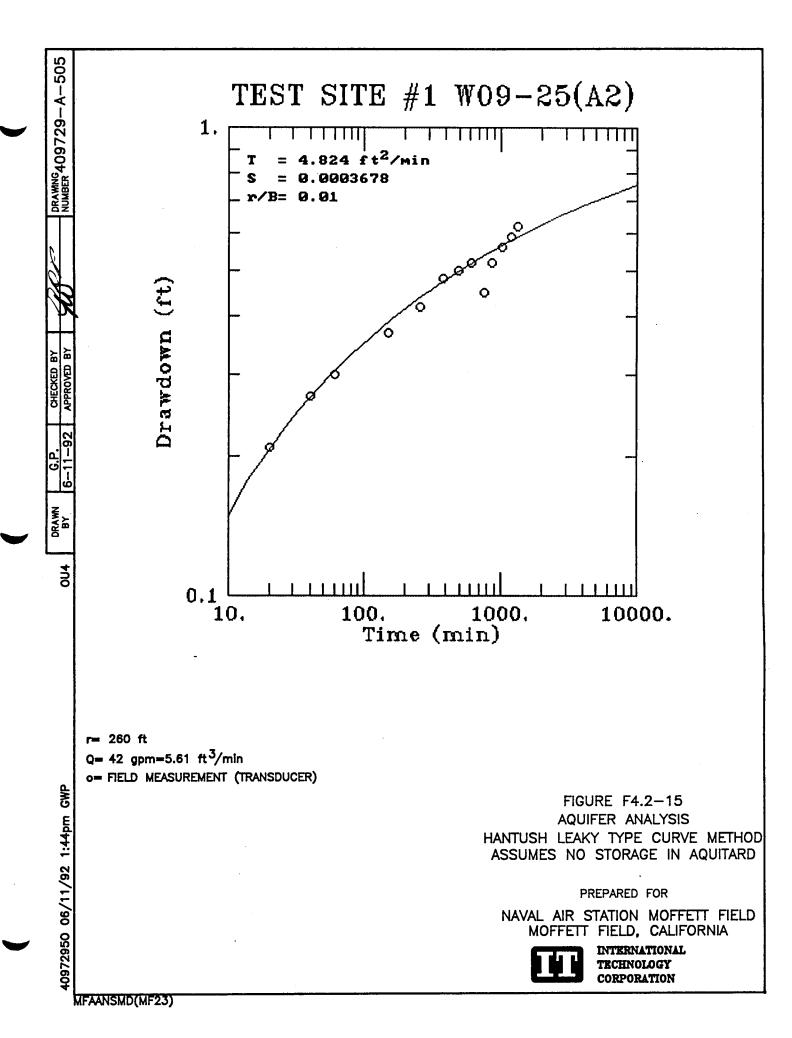
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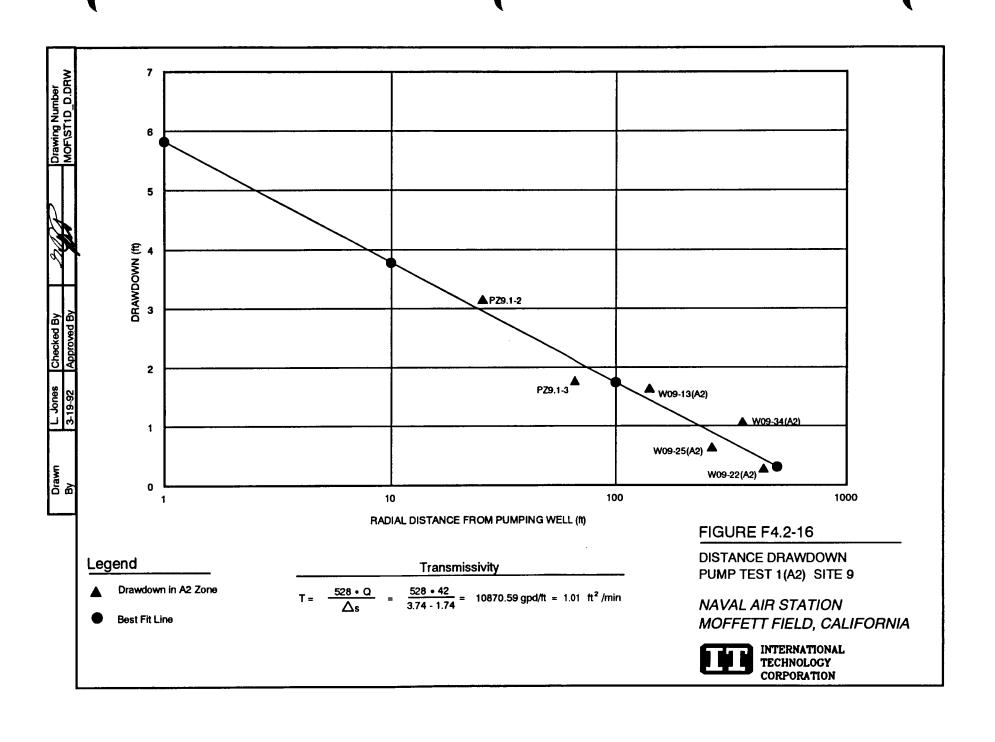




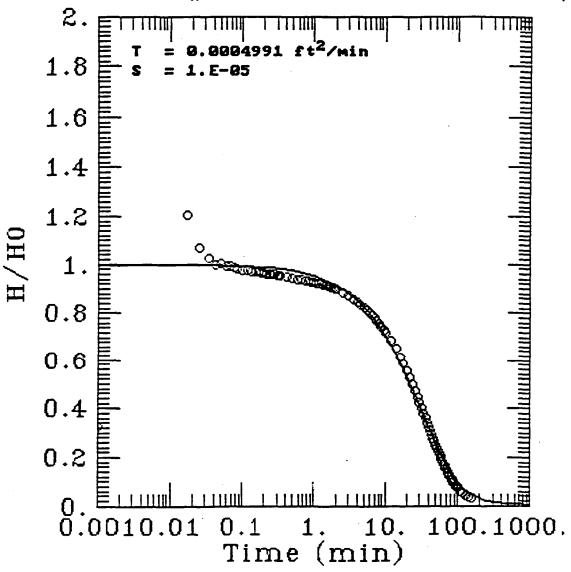








## TEST SITE #1 SLUG OUT PZ9.1-1(AQ)



o= FIELD MEASUREMENT (TRANSDUCER)

USING A UNIT THICKNESS OF 5.5 FT. YIELDS A HORIZONTAL HYDRAULIC CONDUCTIVITY OF 0.000091 FT. 2/MIN.

FIGURE F4.2-17
SLUG TEST ANALYSIS
COOPER, et al. METHOD

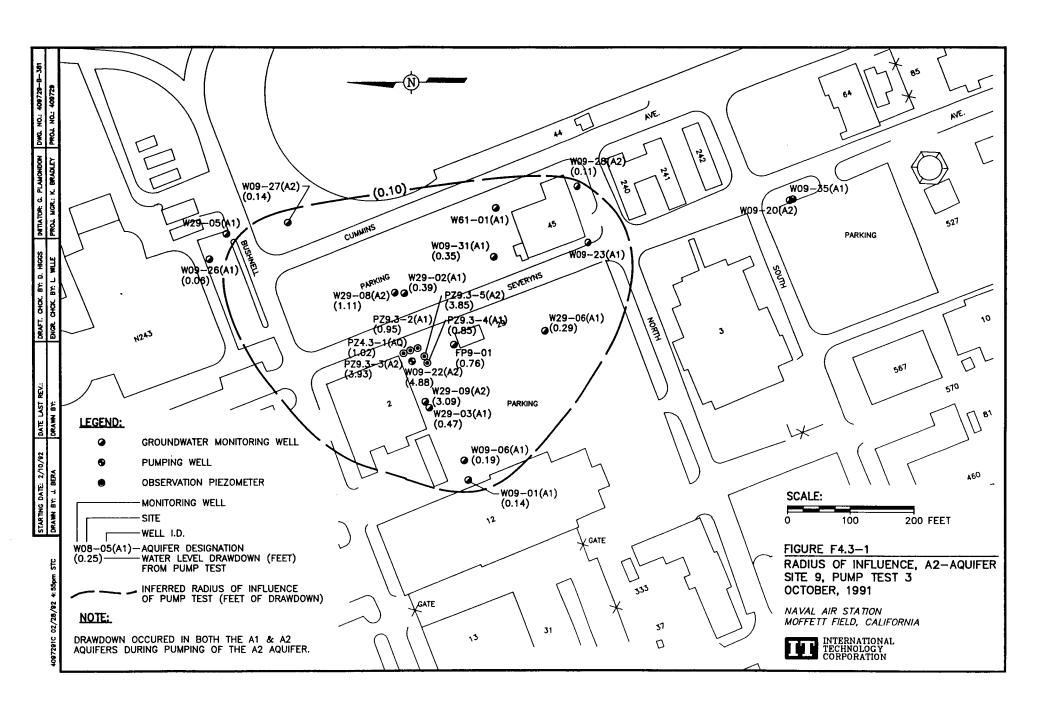
PREPARED FOR

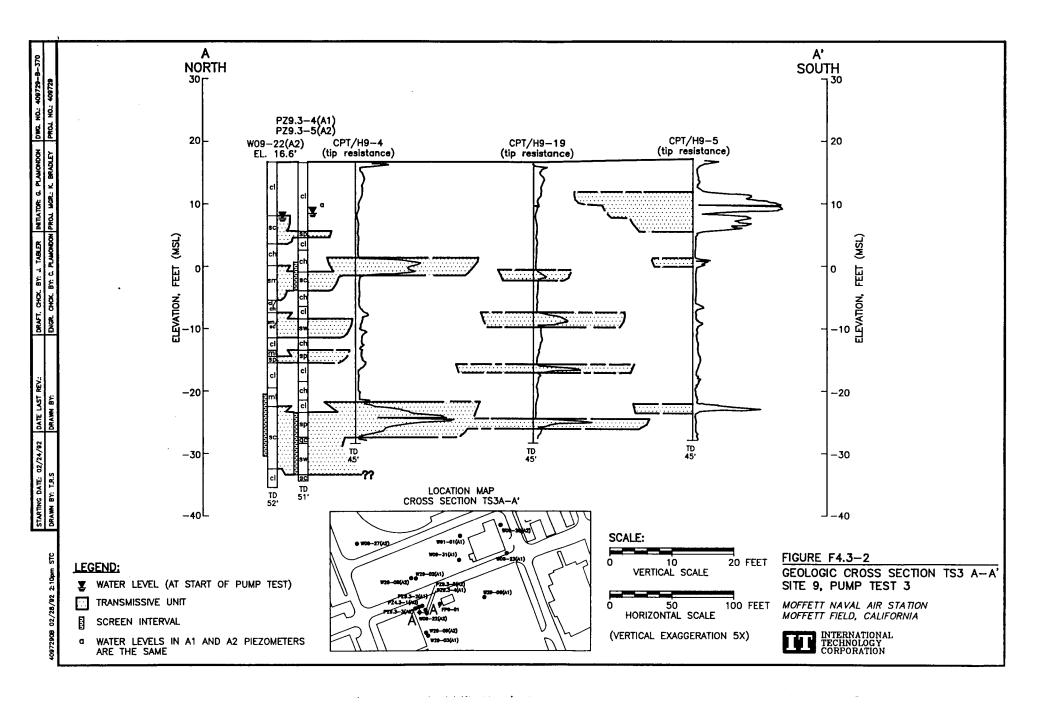
NAVAL AIR STATION MOFFETT FIELD MOFFETT FIELD, CALIFORNIA

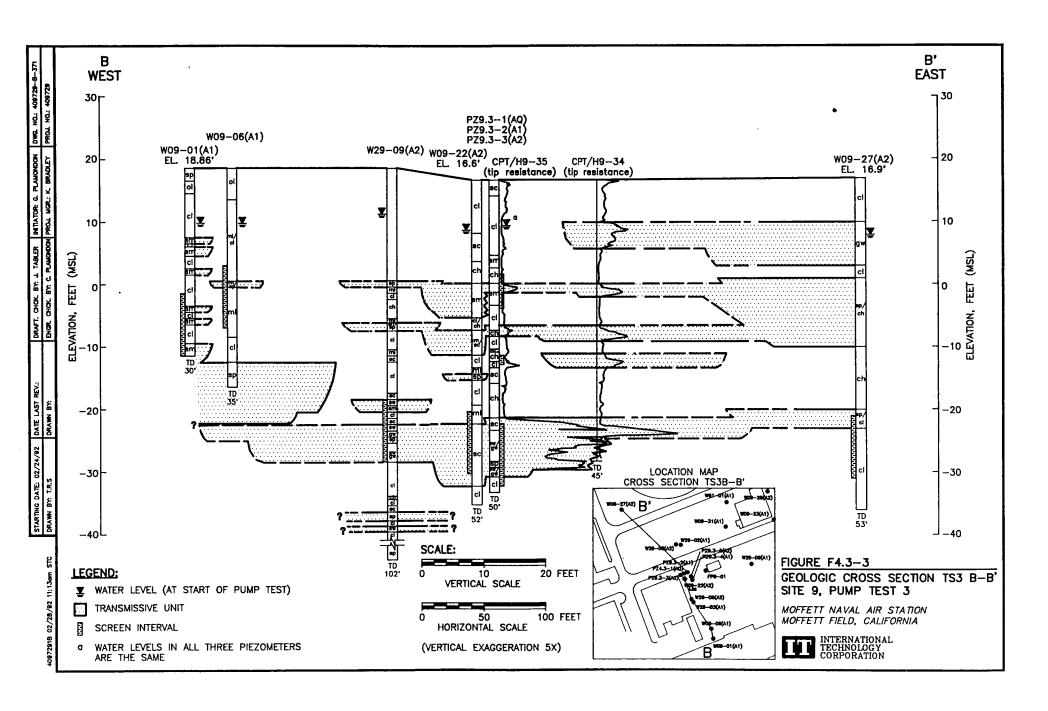


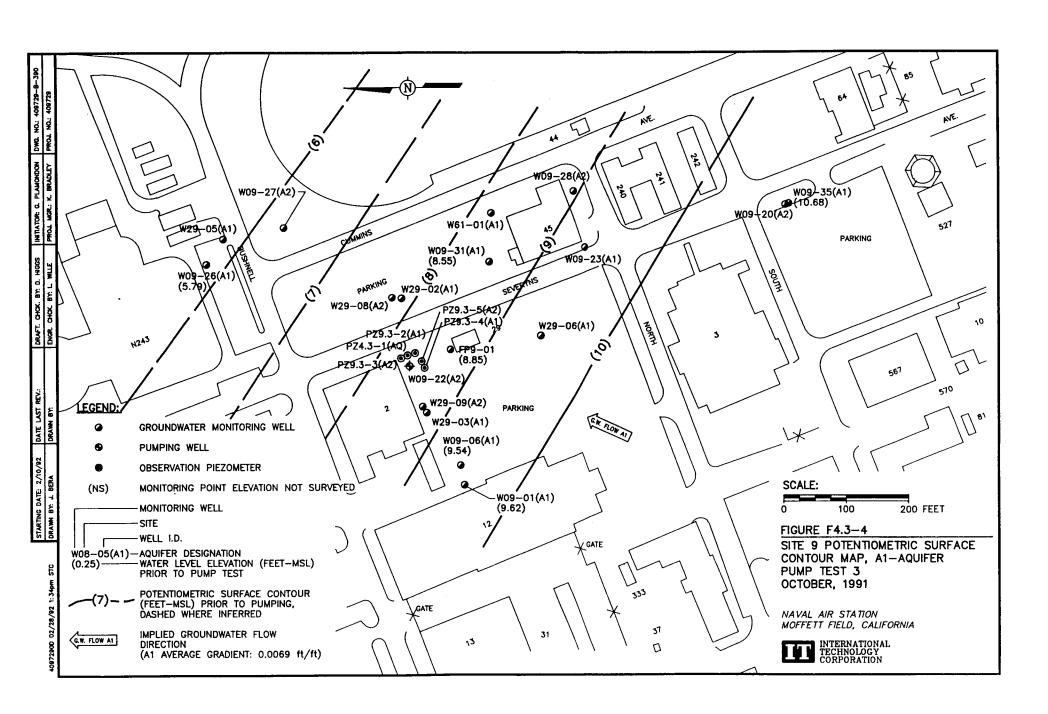
INTERNATIONAL TECHNOLOGY CORPORATION

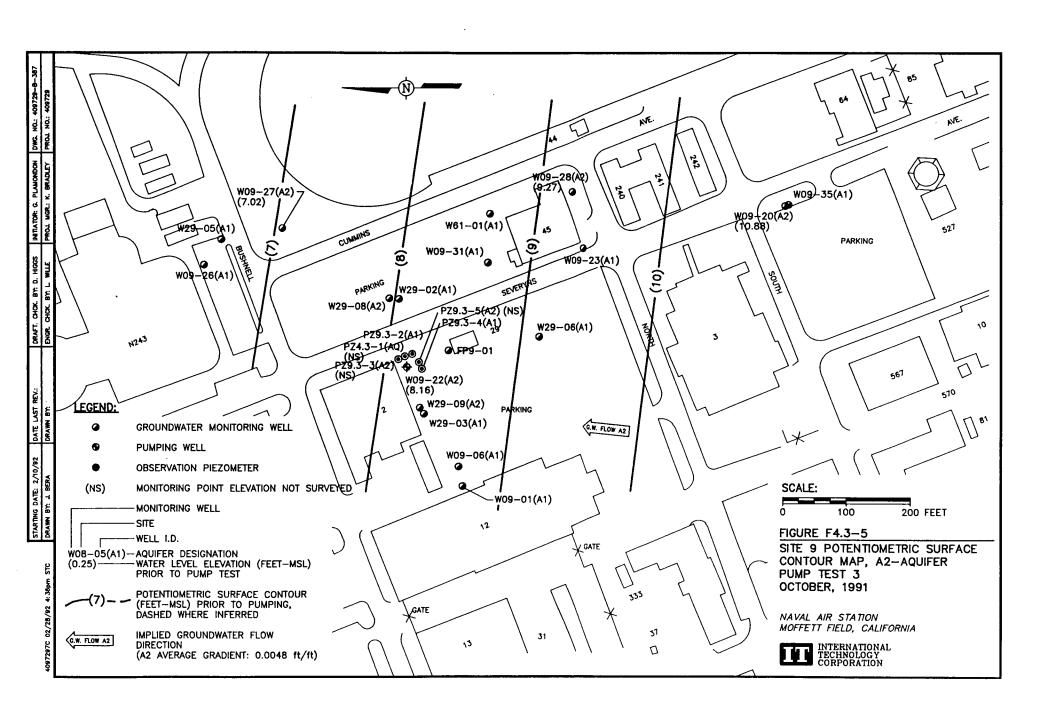
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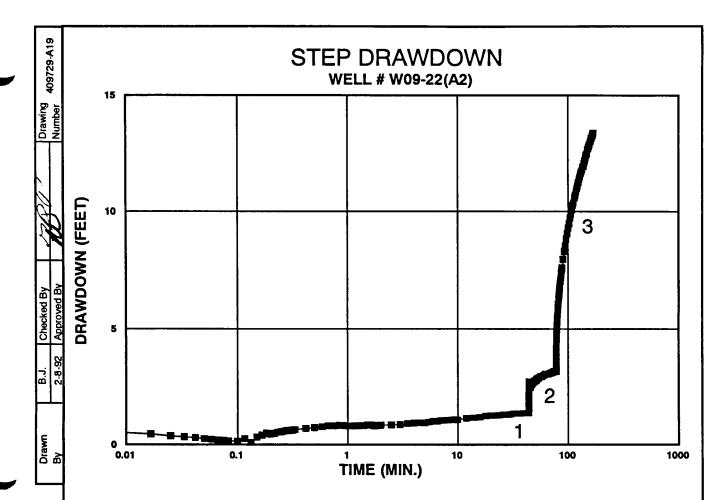












STEP#	DISCHARGE RATE Q (GPM)	CUMULATIVE DRAWDOWN s (feet)	SPECIFIC CAPACITY Q/s (gpm/ft)
1	5	1.45	3.448
2	10	3.65	2.740
3	16.2	13.85	NA

Note:

Stabilization was not achieved for this step due to encroachment on the well screen during the third stage.

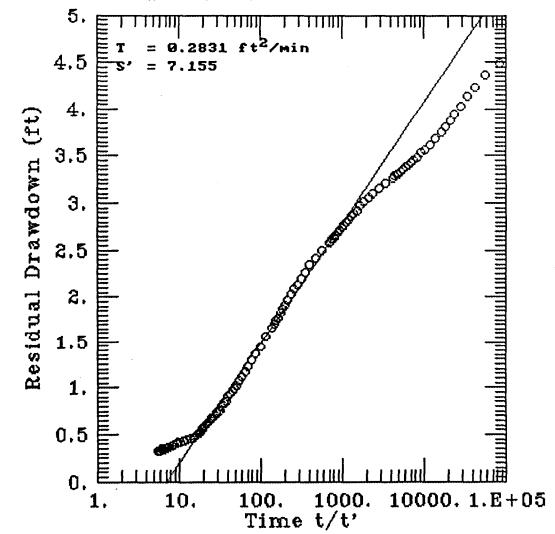
FIGURE F4.3-6

Pump Test 3 Step-Drawdown Test

NAVAL AIR STATION MOFFET FIELD, CALIFORNIA



## TEST SITE #3 (A2) RECOVERY W09-22(A2)



r= 0
Q= 15 gpm=2 ft 3/min
o= FIELD MEASUREMENT (TRANSDUCER)

NOTE:

SINCE LEAKANCE HAS OCCURRED THROUGH THE OVERLYING AQUITARD, A RESTRICTION INCORPORATING THE LEAKAGE FACTOR IS DICTATED BY:

 $tp + t' < (B^2S)/20T$ 

THEREFORE, VALUES OF T MAY BE OVERESTIMATED.

FIGURE F4.3-7
AQUIFER ANALYSIS
THEIS RECOVERY METHOD

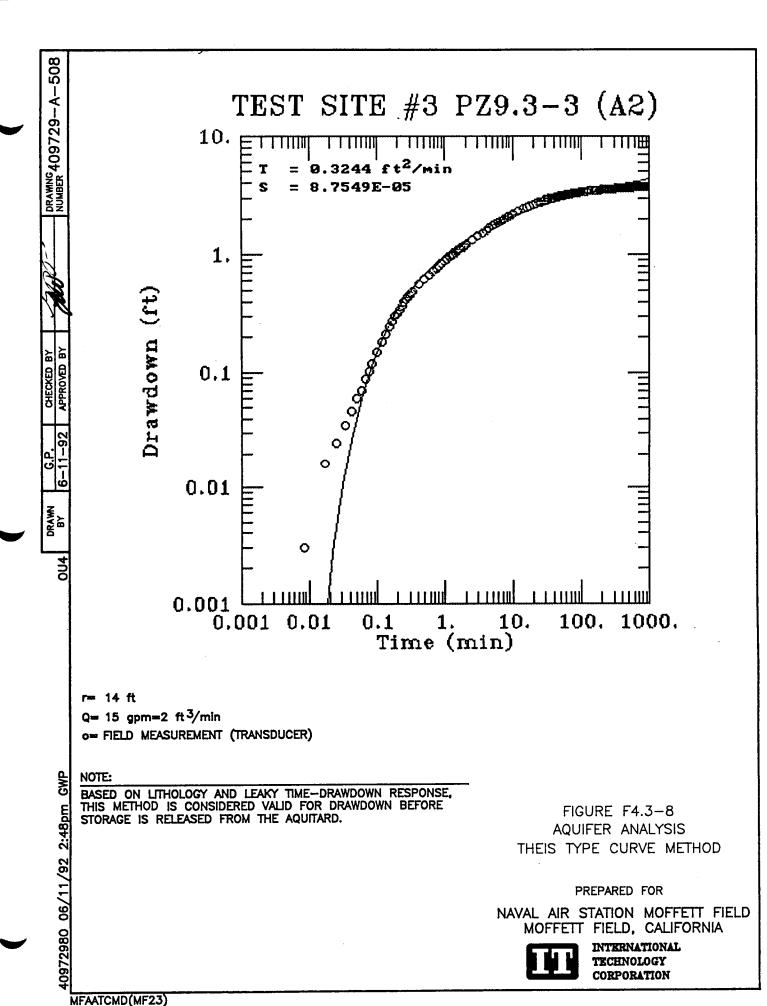
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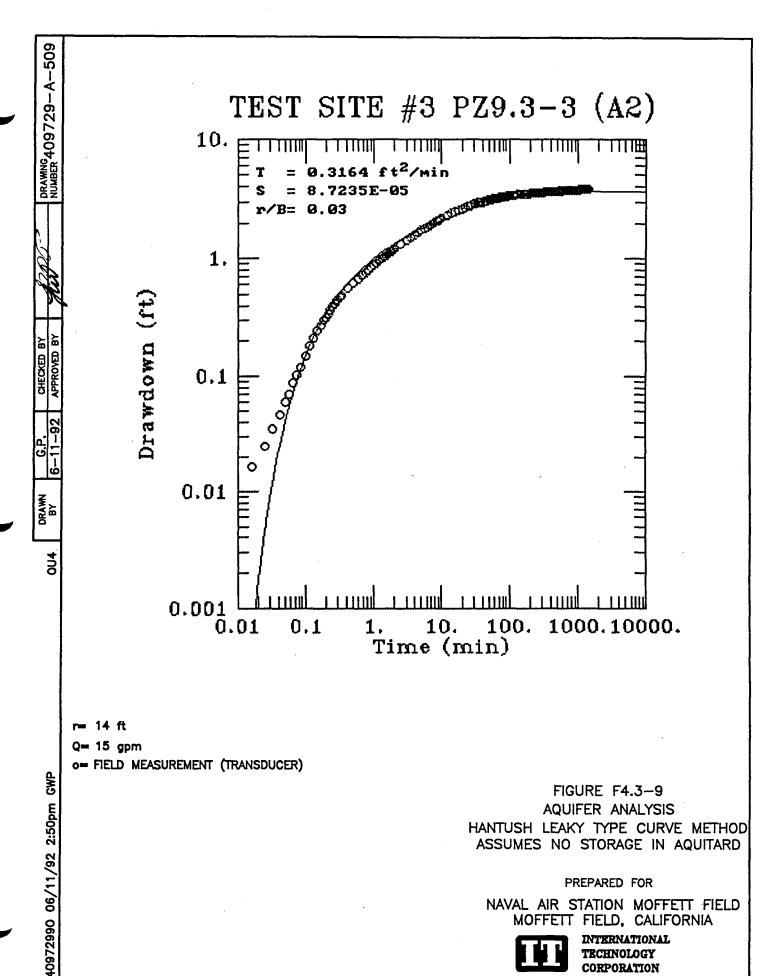
NAVAL AIR STATION MOFFETT FIELD MOFFETT FIELD, CALIFORNIA



INTERNATIONAL TECHNOLOGY CORPORATION

72970 06/15/92 6:06pm GWP





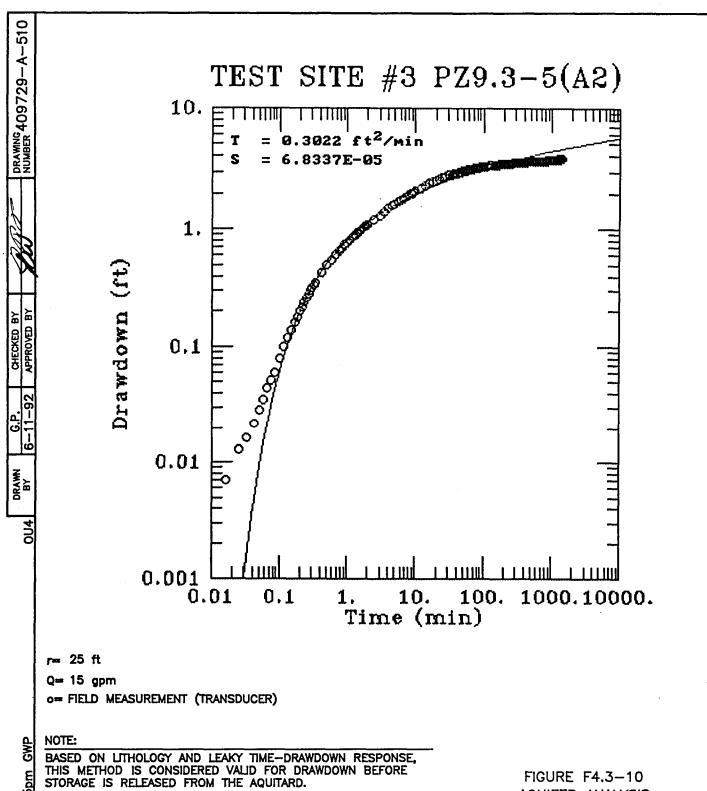


FIGURE F4.3-10
AQUIFER ANALYSIS
THEIS TYPE CURVE METHOD

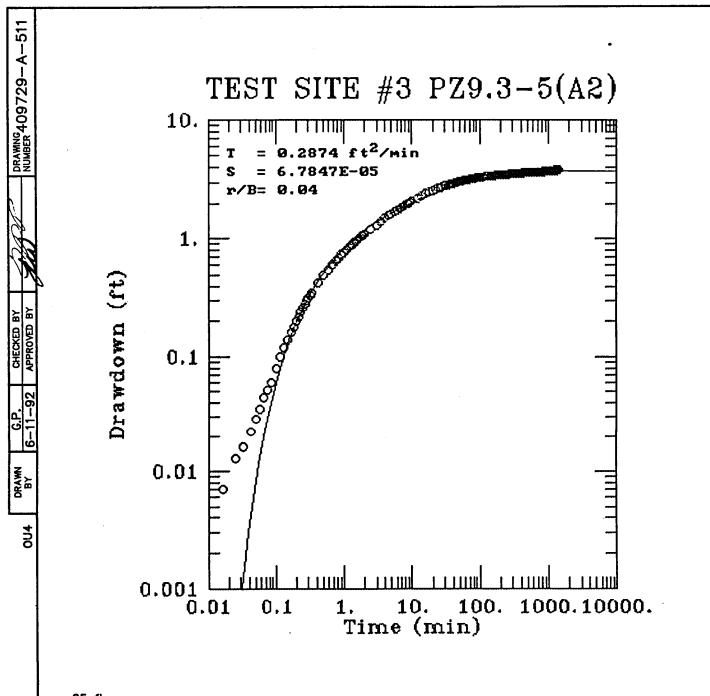
PREPARED FOR

NAVAL AIR STATION MOFFETT FIELD MOFFETT FIELD, CALIFORNIA



INTERNATIONAL TECHNOLOGY CORPORATION

MFAATCMD(MF23)



r= 25 ft

Q= 15 gpm=2 ft 3/min

o= FIELD MEASUREMENT (TRANSDUCER)

FIGURE F4.3-11

AQUIFER ANALYSIS

HANTUSH LEAKY TYPE CURVE METHOD

ASSUMES NO STORAGE IN AQUITARD

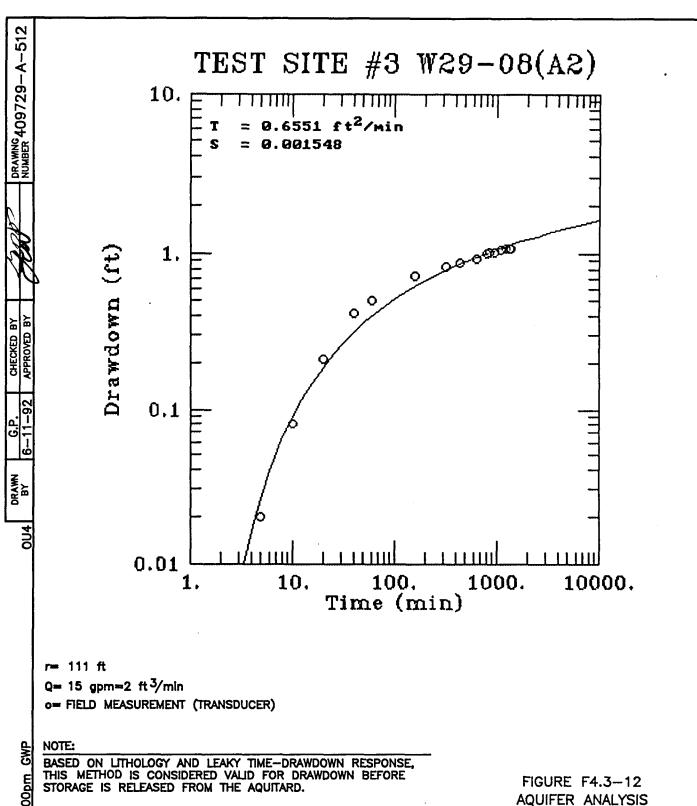
PREPARED FOR

NAVAL AIR STATION MOFFETT FIELD MOFFETT FIELD, CALIFORNIA



INTERNATIONAL TECHNOLOGY CORPORATION

4097291P 06/11/92 2:57pm GWF



THEIS TYPE CURVE METHOD

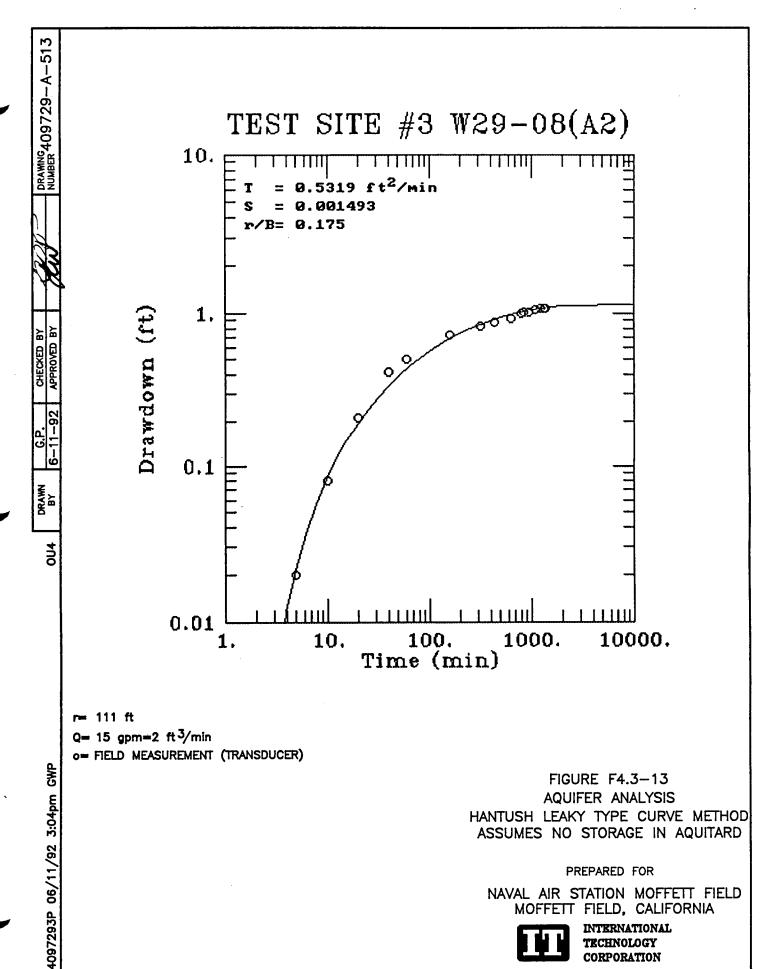
PREPARED FOR

NAVAL AIR STATION MOFFETT FIELD MOFFETT FIELD, CALIFORNIA

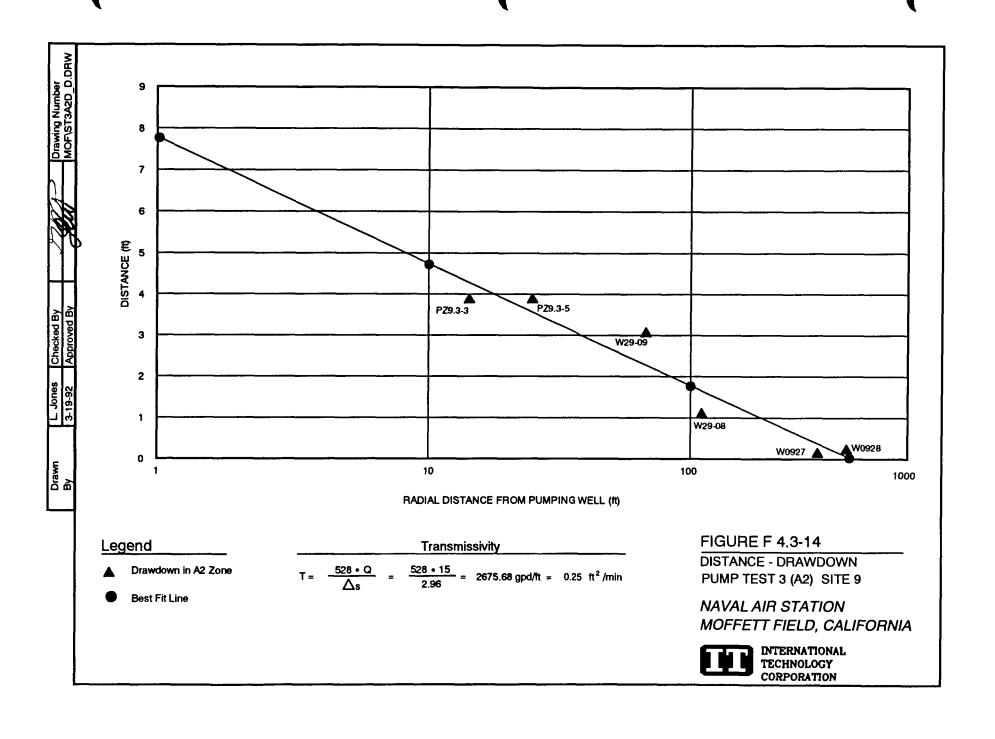


INTERNATIONAL TECHNOLOGY CORPORATION

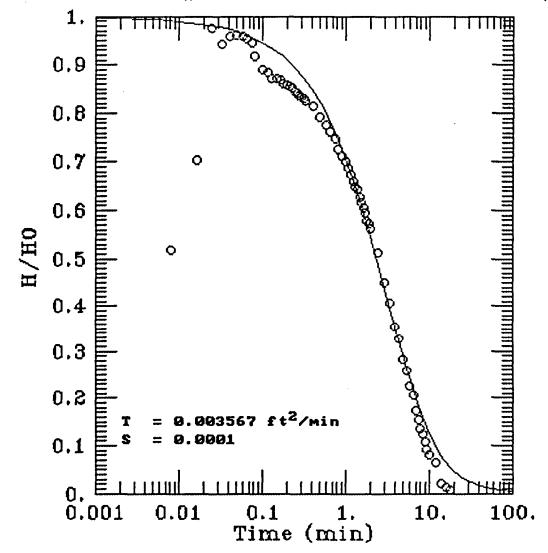
MFAATCMD(MF23)



MFAANSMD(MF23)



## TEST SITE #3 SLUG OUT PZ9.3-1(AQ)



o= FIELD MEASUREMENT (TRANSDUCER)

USING A UNIT THICKNESS OF 17.0 FT. YIELDS A HORIZONTAL HYDRAULIC CONDUCTIVITY OF 0.00023 FT. 2/MIN.

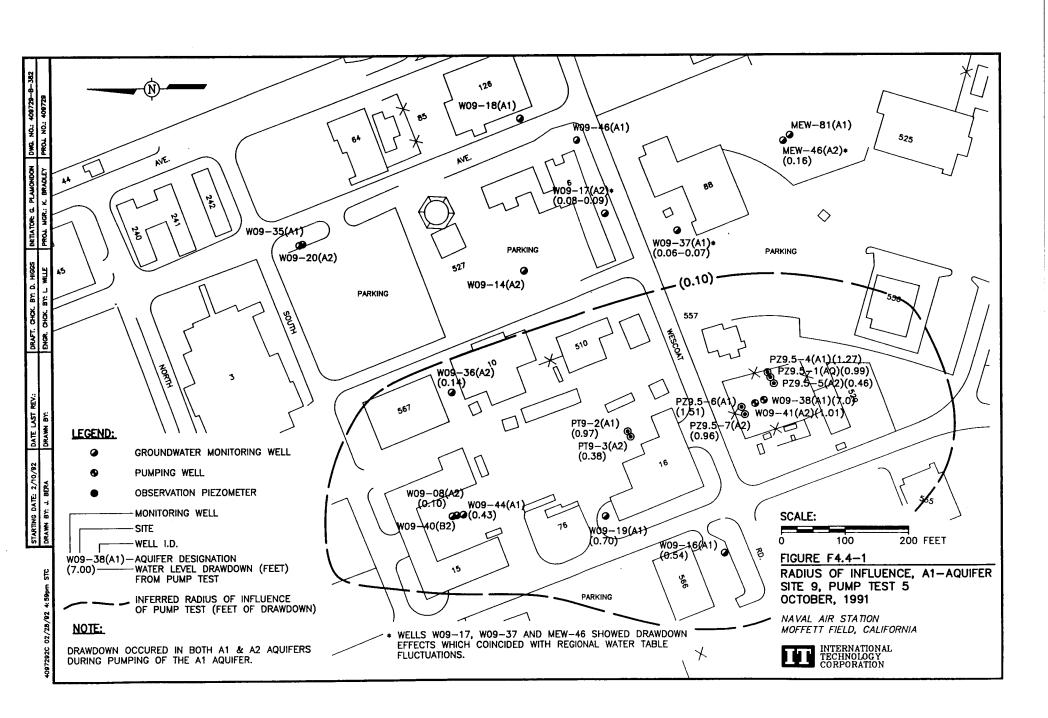
FIGURE F4.3-15 SLUG TEST ANALYSIS COOPER, et al. METHOD

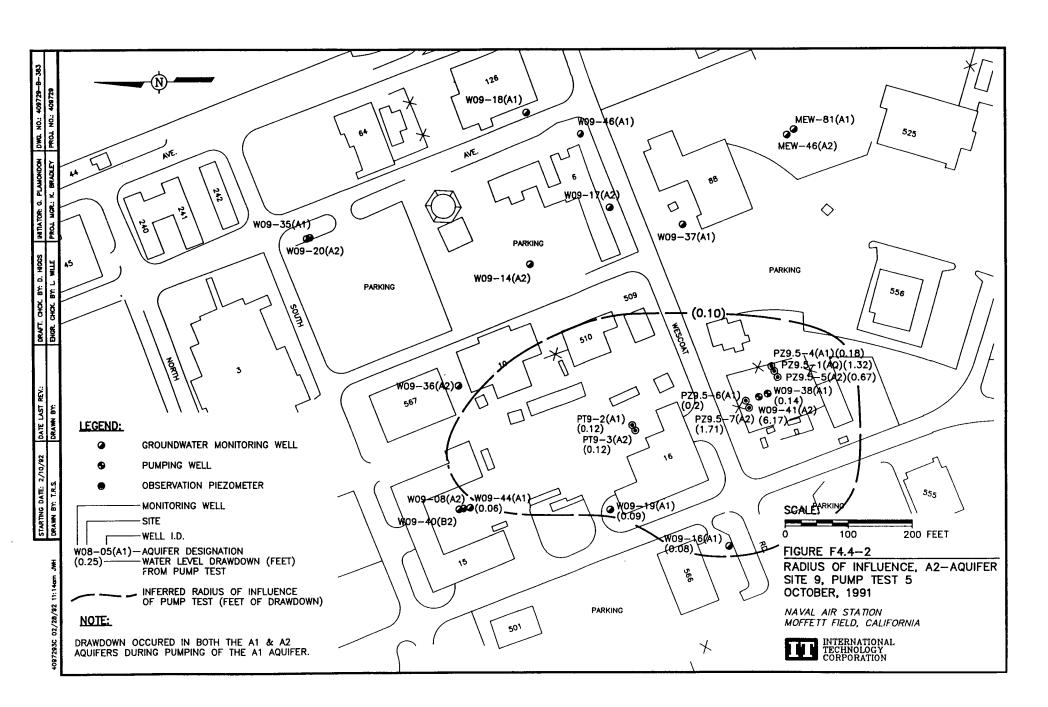
PREPARED FOR

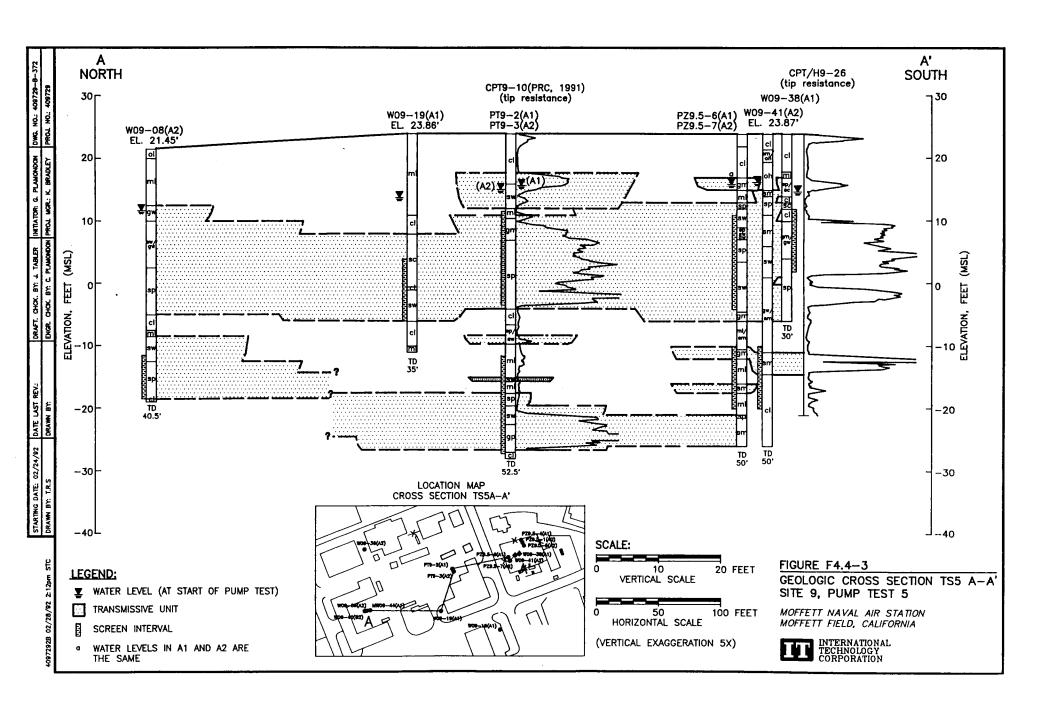
NAVAL AIR STATION MOFFETT FIELD MOFFETT FIELD, CALIFORNIA

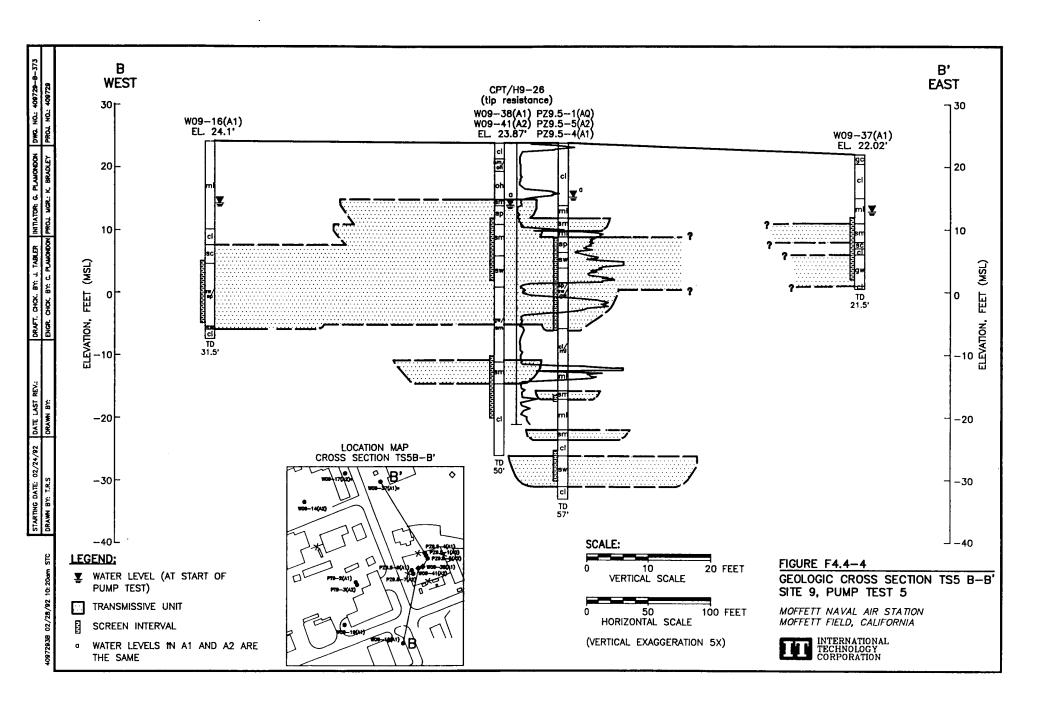


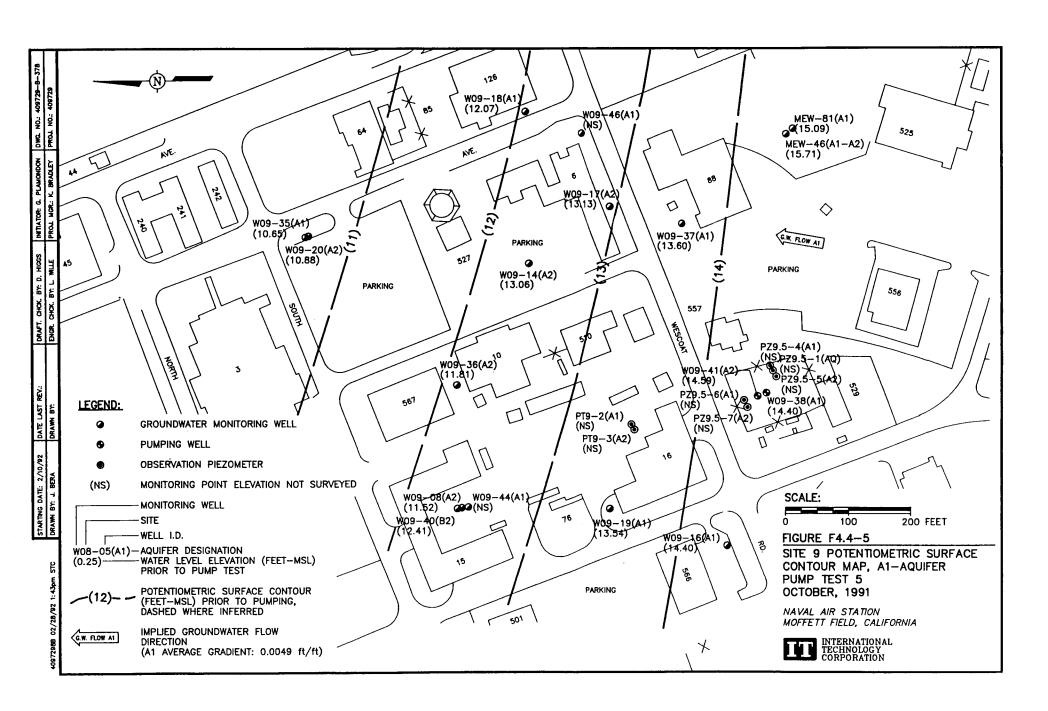
INTERNATIONAL TECHNOLOGY CORPORATION

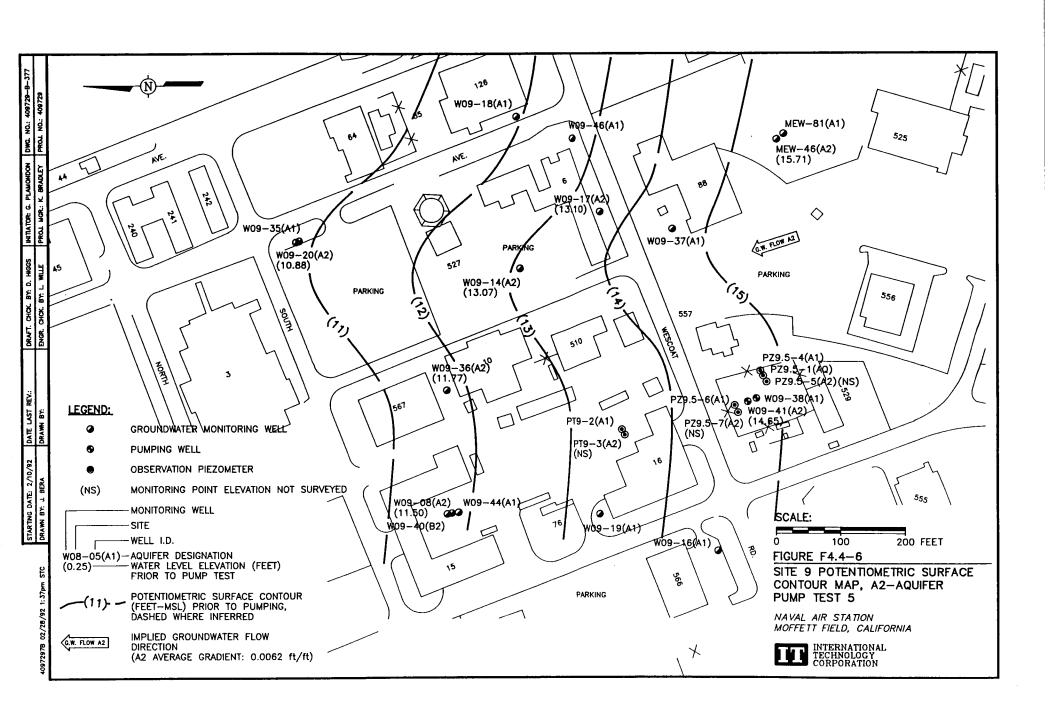


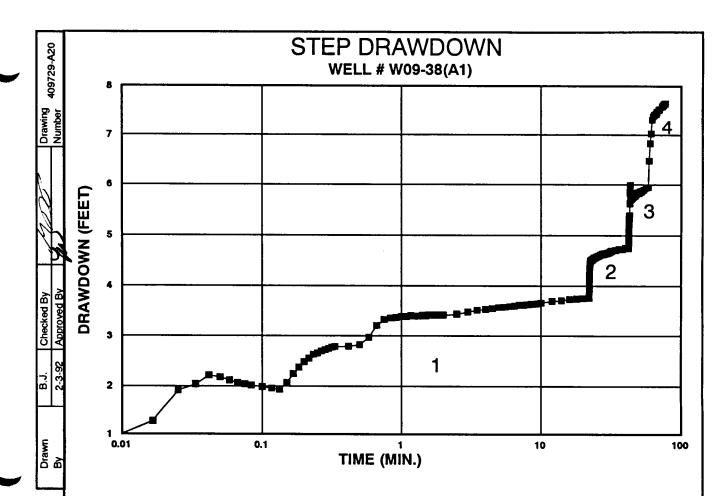












STEP#	DISCHARGE RATE Q (GPM)	CUMULATIVE DRAWDOWN s (feet)	SPECIFIC CAPACITY Q/s (gpm/ft)
1	31	3.75	8.267
2	37	4.75	7.789
3	41.5	5.9	7.034
4	50	7.85	6.369

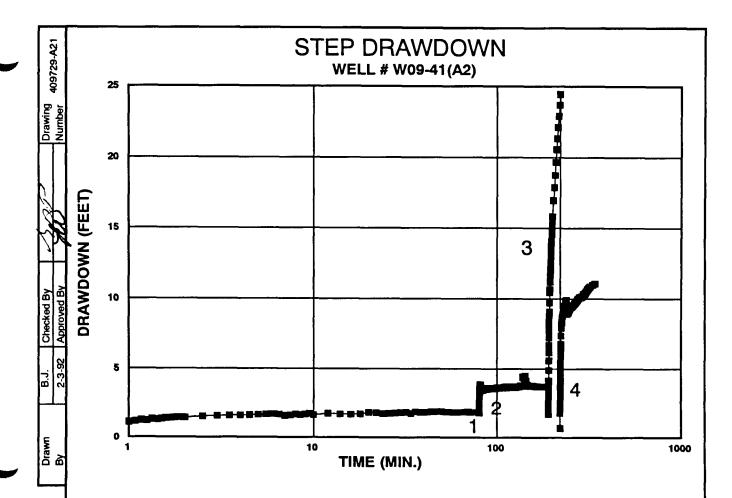
## Note:

The initial step-drawdown test included discharge rates ranging from 3.4 gpm to 31 gpm and is not included in the graphical analysis of well W09-38(A1).

FIGURE F4.4-7 Pump Test 5 (A1) Step-Drawdown Test

NAVAL AIR STATION MOFFET FIELD, CALIFORNIA





STEP#	DISCHARGE RATE Q (GPM)	CUMULATIVE DRAWDOWN s (feet)	SPECIFIC CAPACITY Q/s (gpm/ft)
1	2	2	1.000
2	4	4	1.000
3	8	>23	*
4	5.4	12.2	0.443

Note:

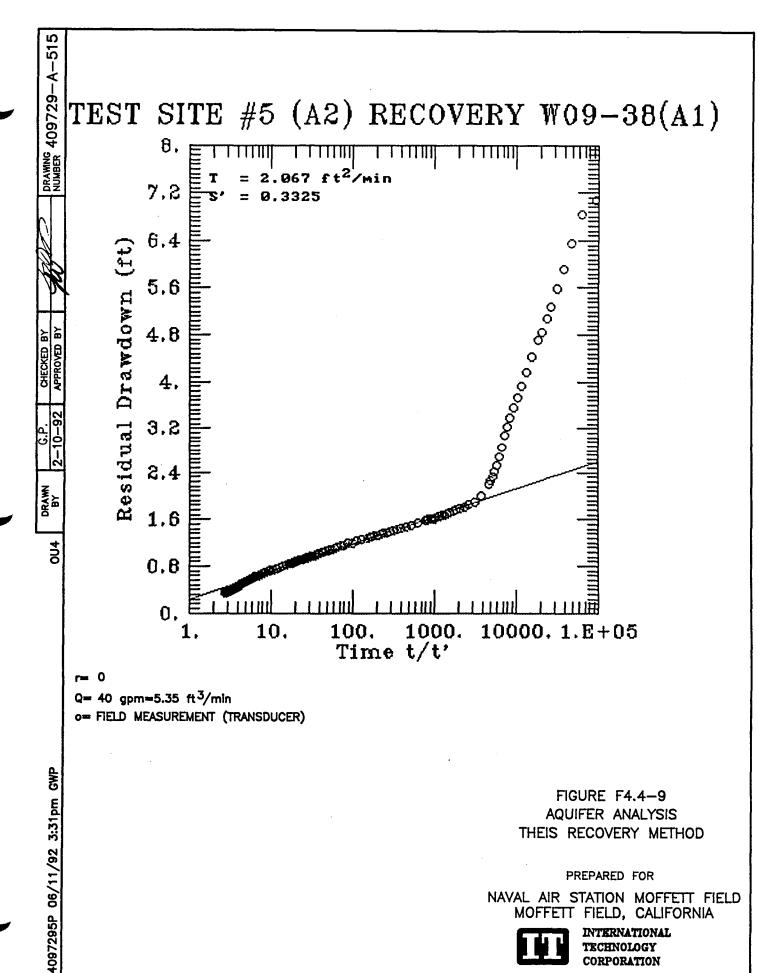
During the third step encroachment of the water level to the well screen occurred and the discharge rate was reduced.

FIGURE F4.4-8

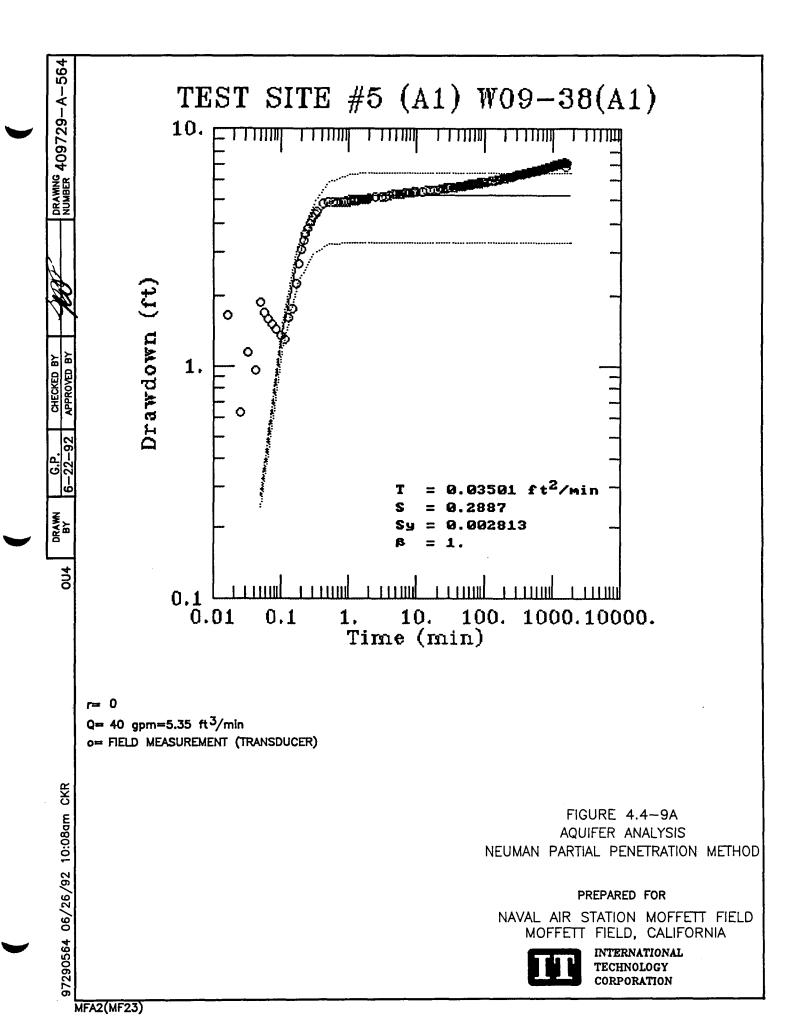
Pump Test 5 (A2) Step-Drawdown Test

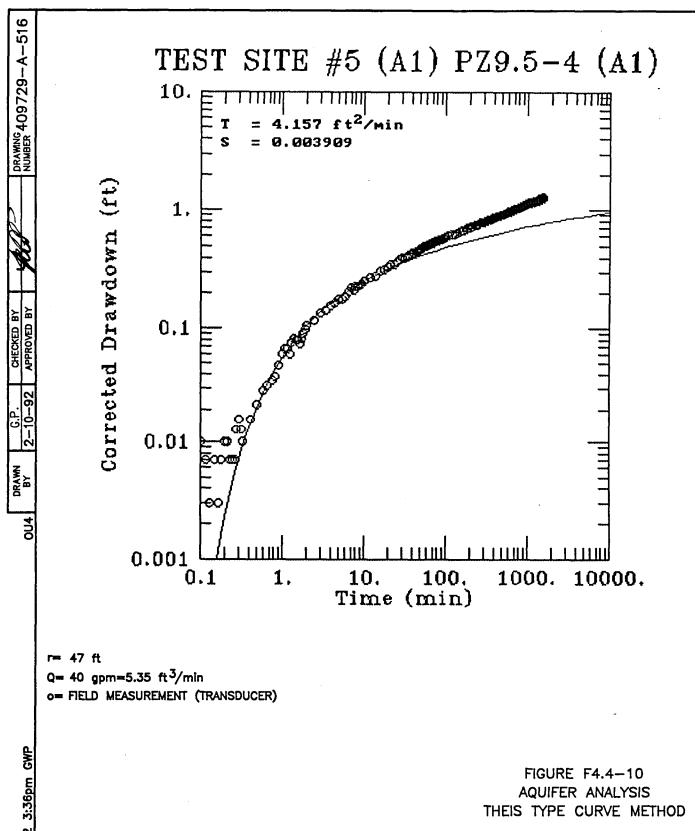
NAVAL AIR STATION MOFFET FIELD, CALIFORNIA





MFA2(MF23)





PREPARED FOR

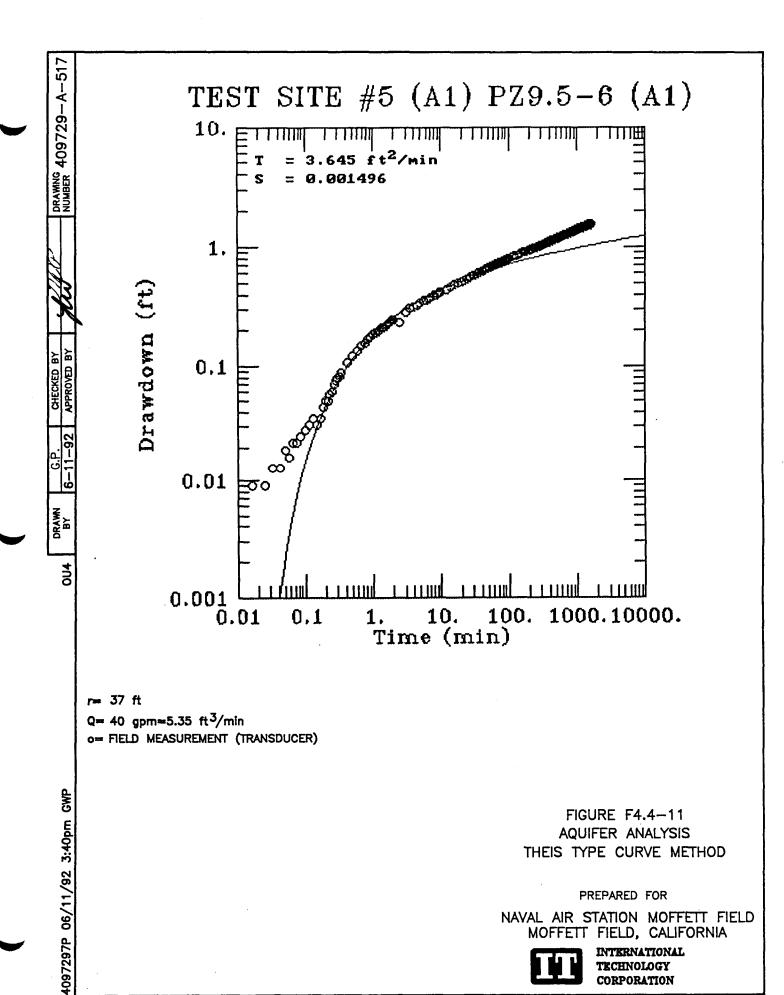
NAVAL AIR STATION MOFFETT FIELD MOFFETT FIELD, CALIFORNIA



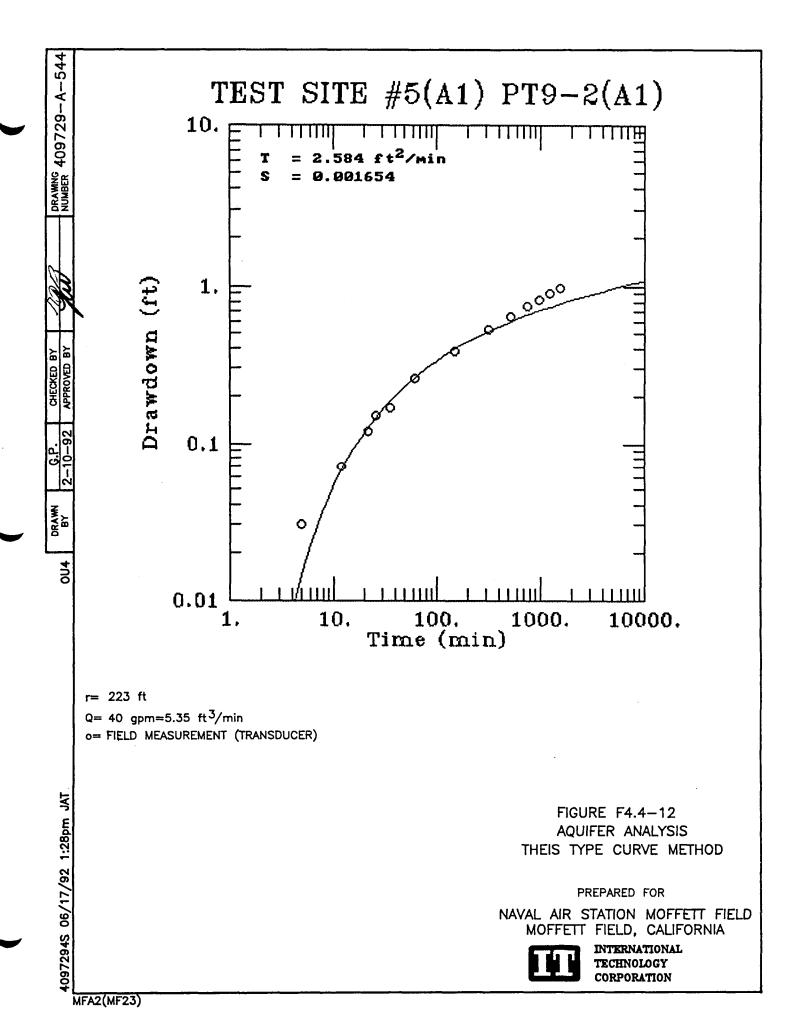
INTERNATIONAL TECHNOLOGY CORPORATION

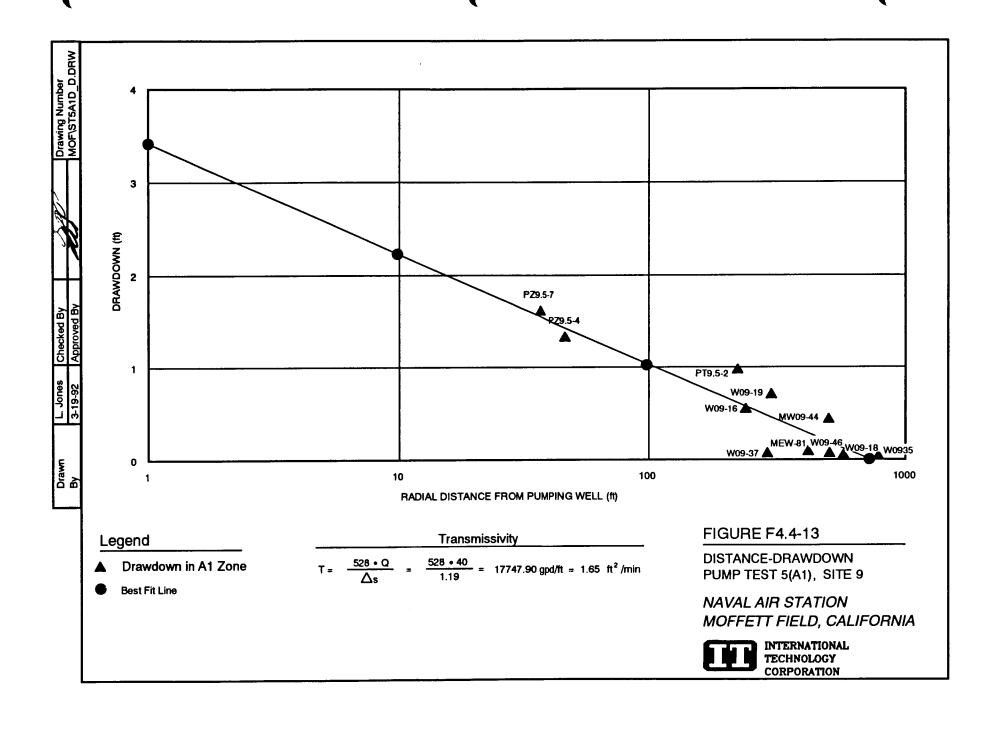
1097296P 06/11/90 3:36pm

MFA1(MF23)



MFA2(MF23)





SINCE LEAKANCE HAS OCCURRED THROUGH THE OVERLYING AQUITARD, A RESTRICTION INCORPORATING THE LEAKAGE FACTOR IS DICTATED BY:

 $tp + t' < (B^2S)/20T$ 

THEREFORE, VALUES OF T MAY BE OVERESTIMATED.

FIGURE F4.4-14
AQUIFER ANALYSIS
THEIS RECOVERY METHOD

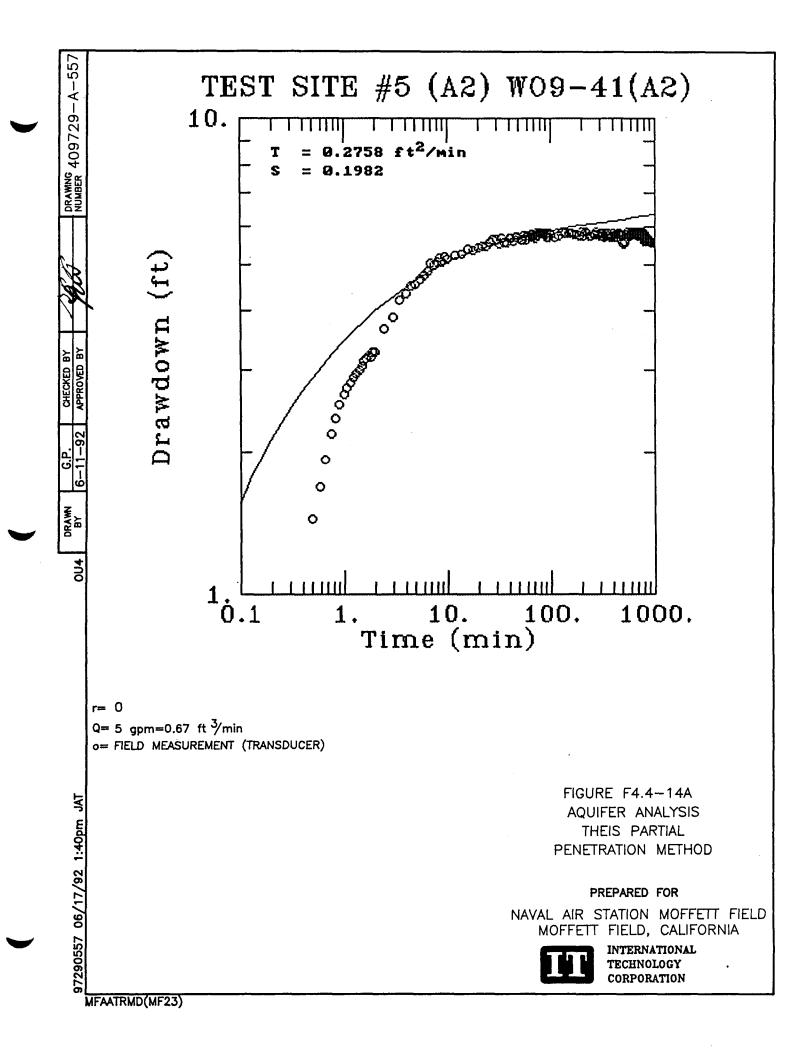
PREPARED FOR

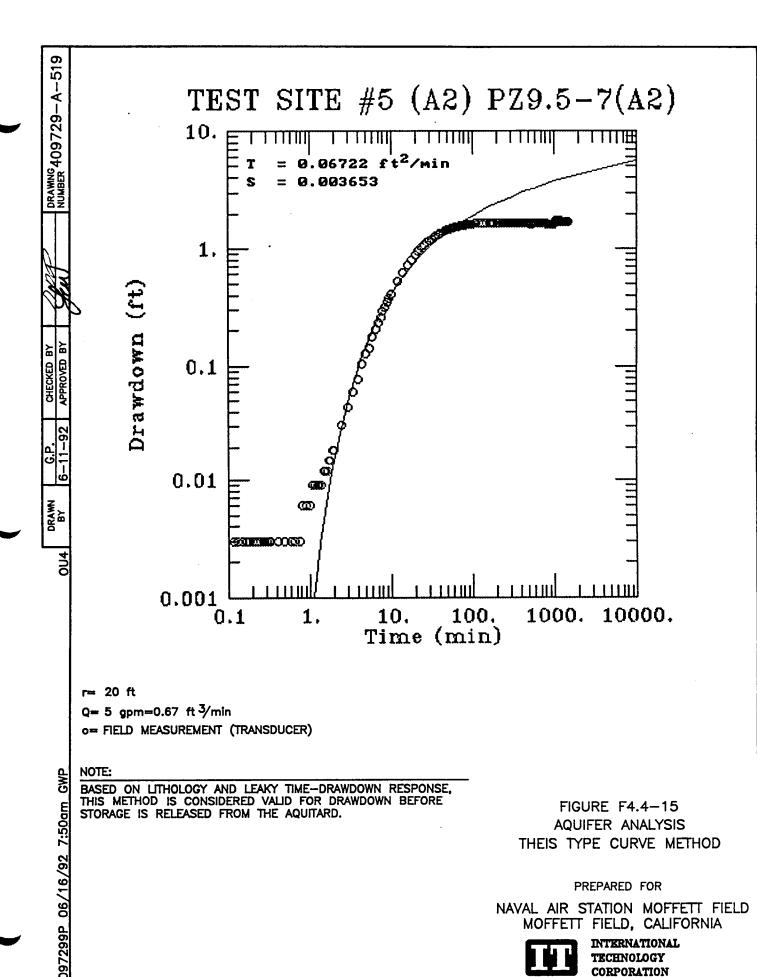
NAVAL AIR STATION MOFFETT FIELD MOFFETT FIELD, CALIFORNIA



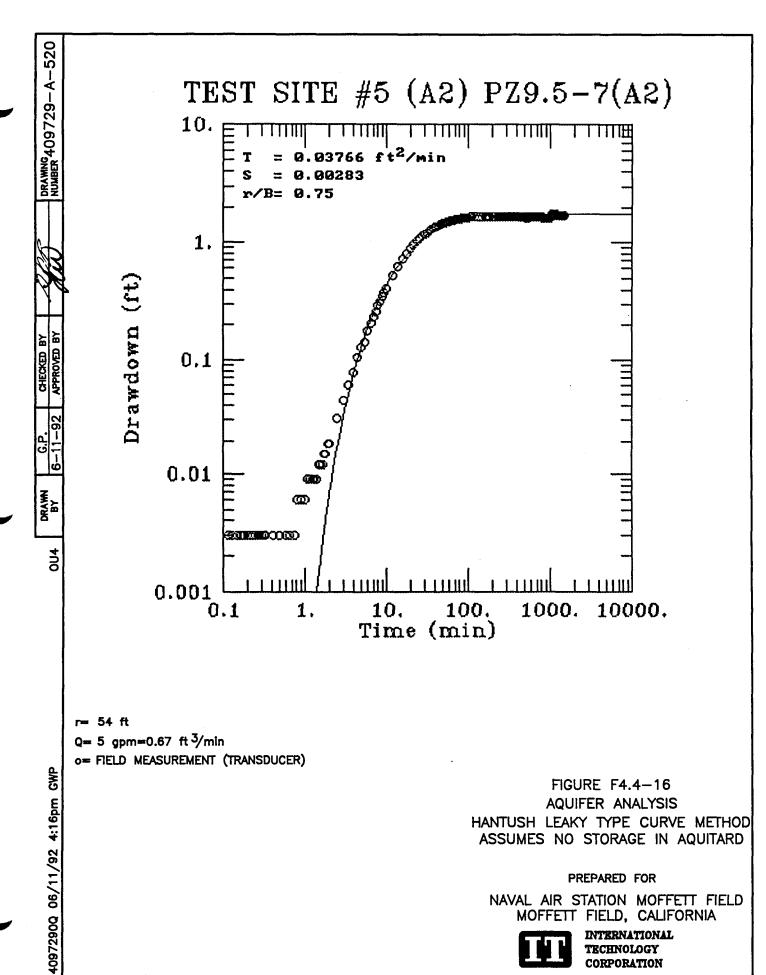
INTERNATIONAL TECHNOLOGY CORPORATION

1097298P 06/15/92 6:18pm GWF

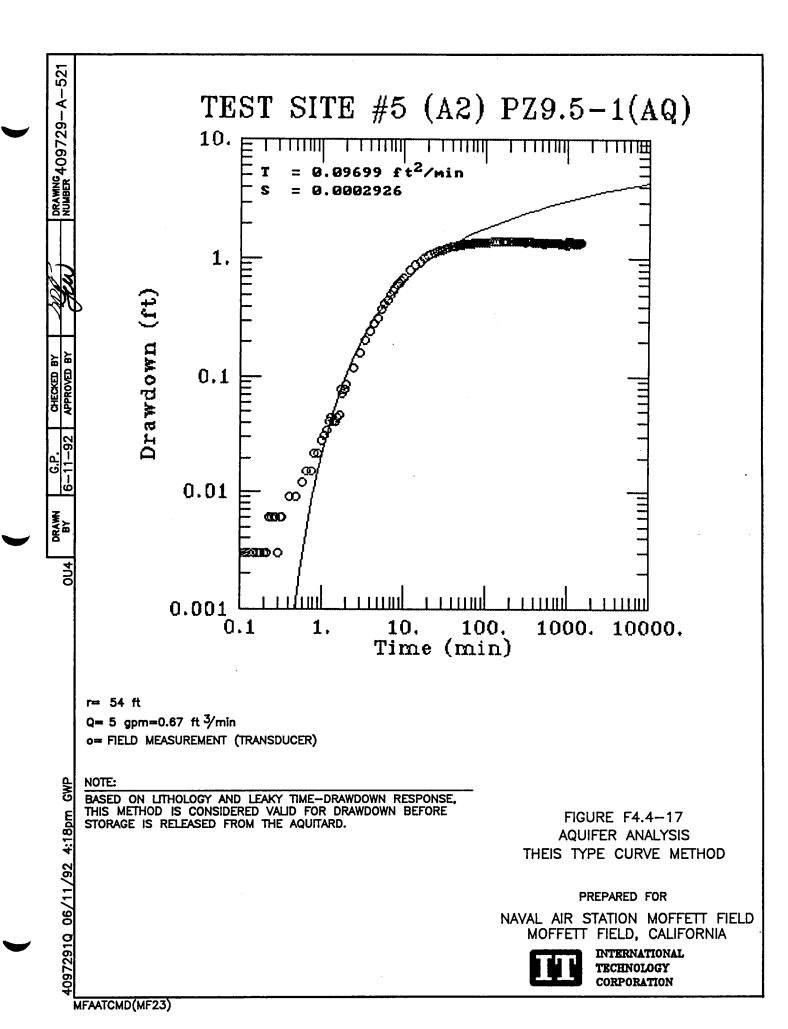


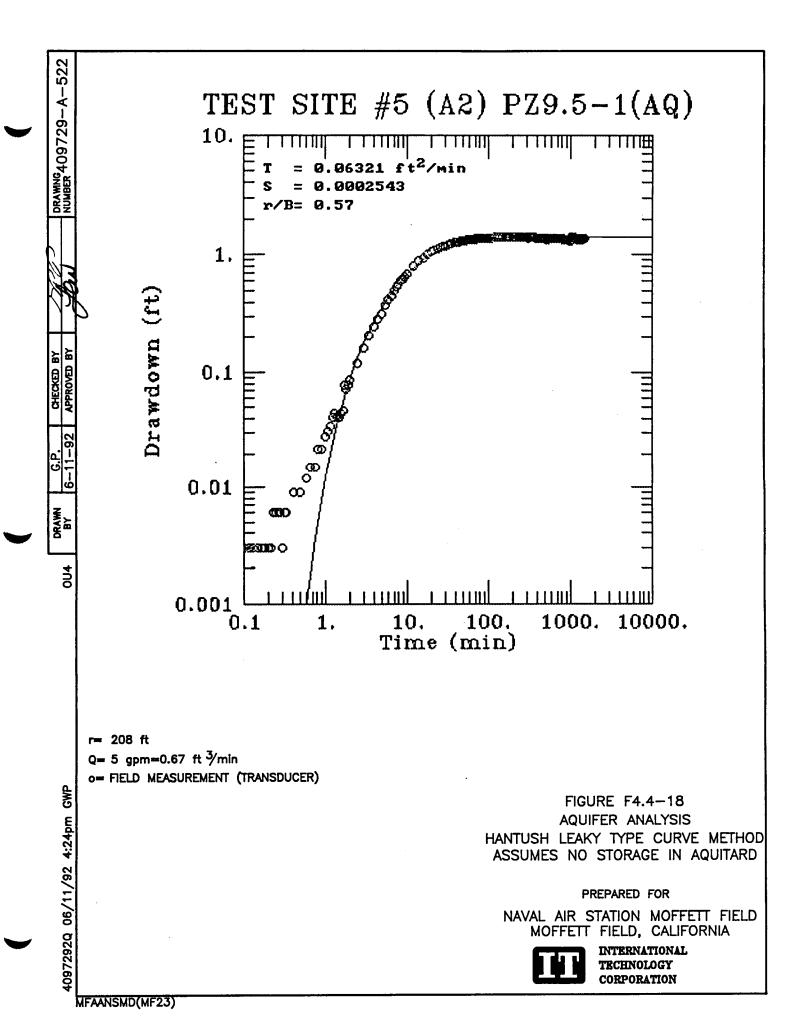


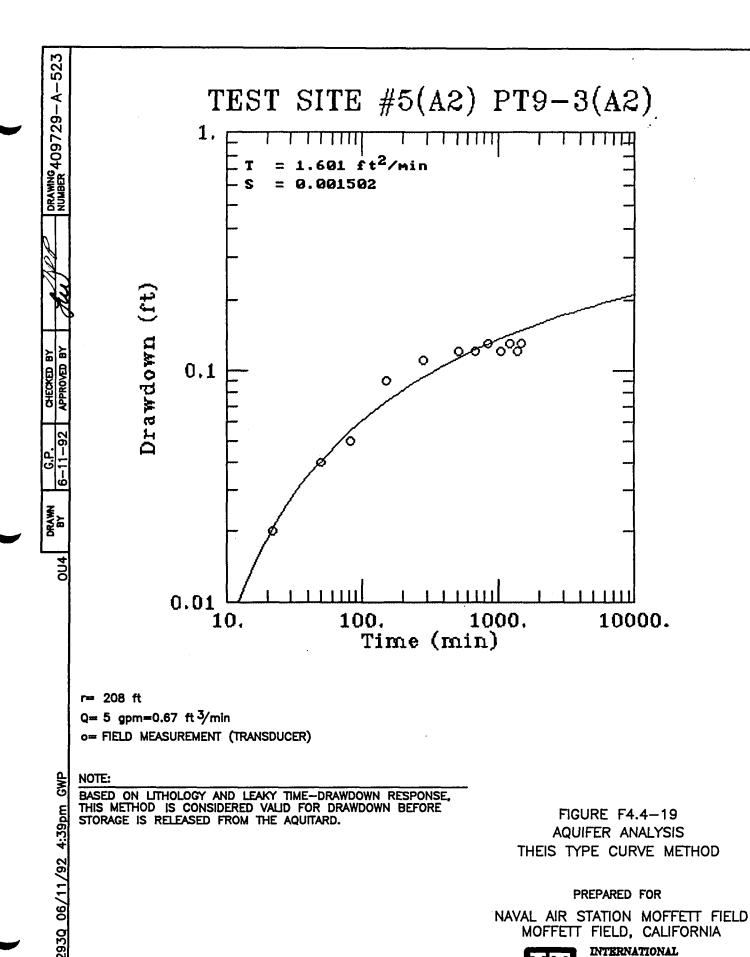
MFAATCMD(MF23)



MFAANSMD(MF23)

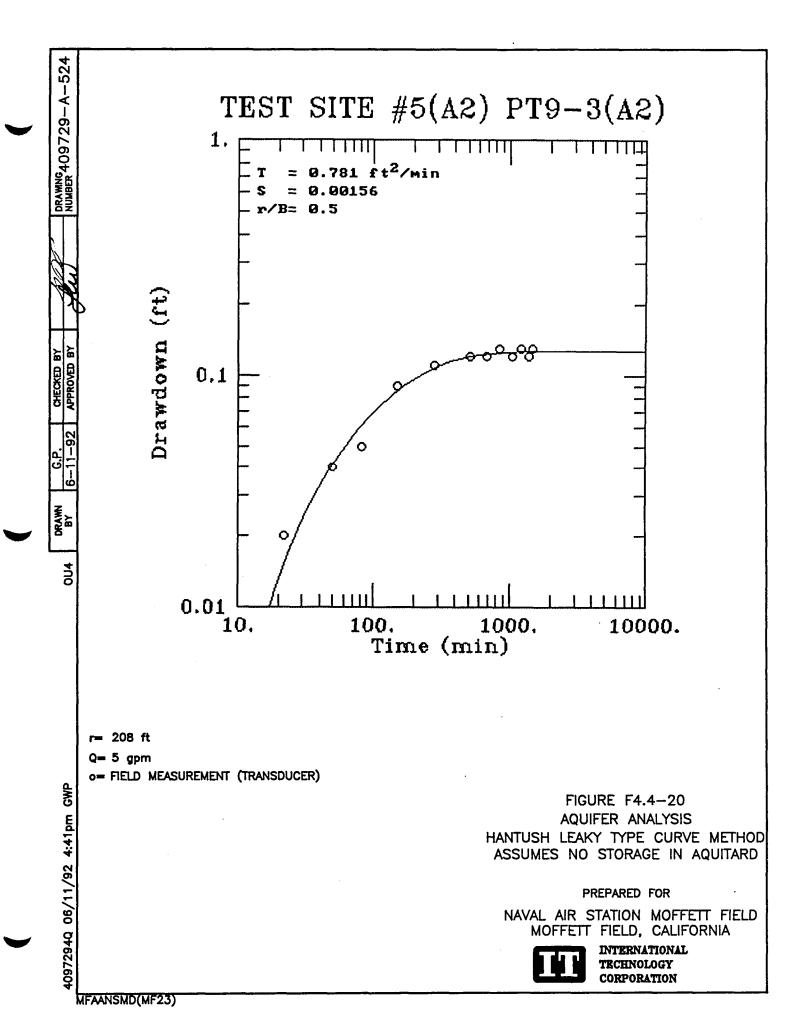


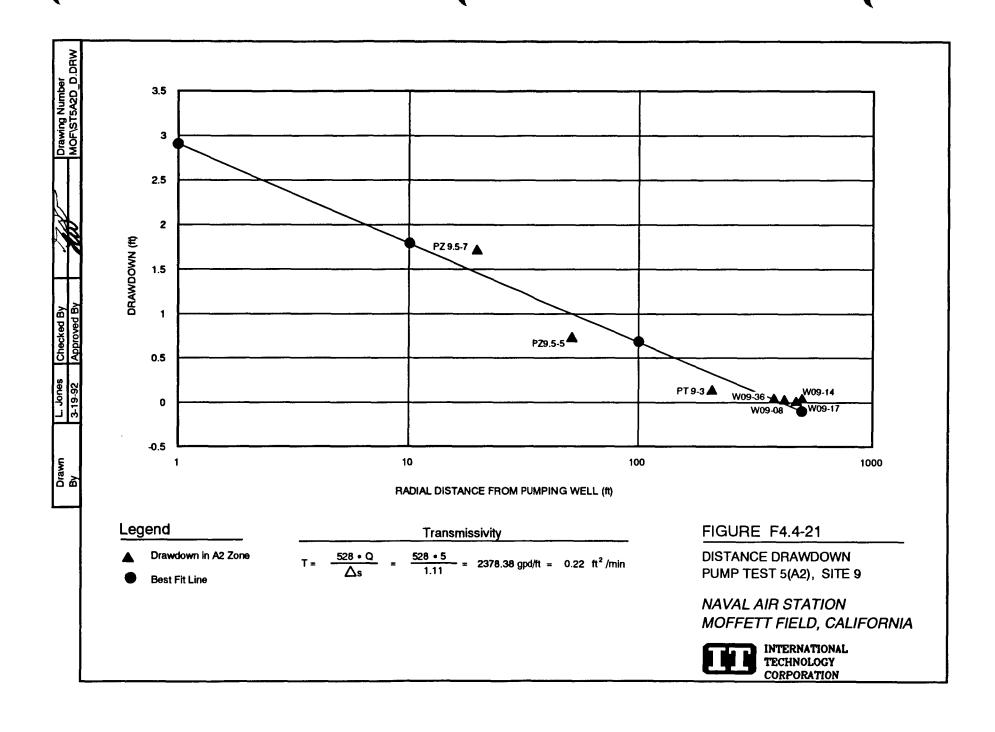


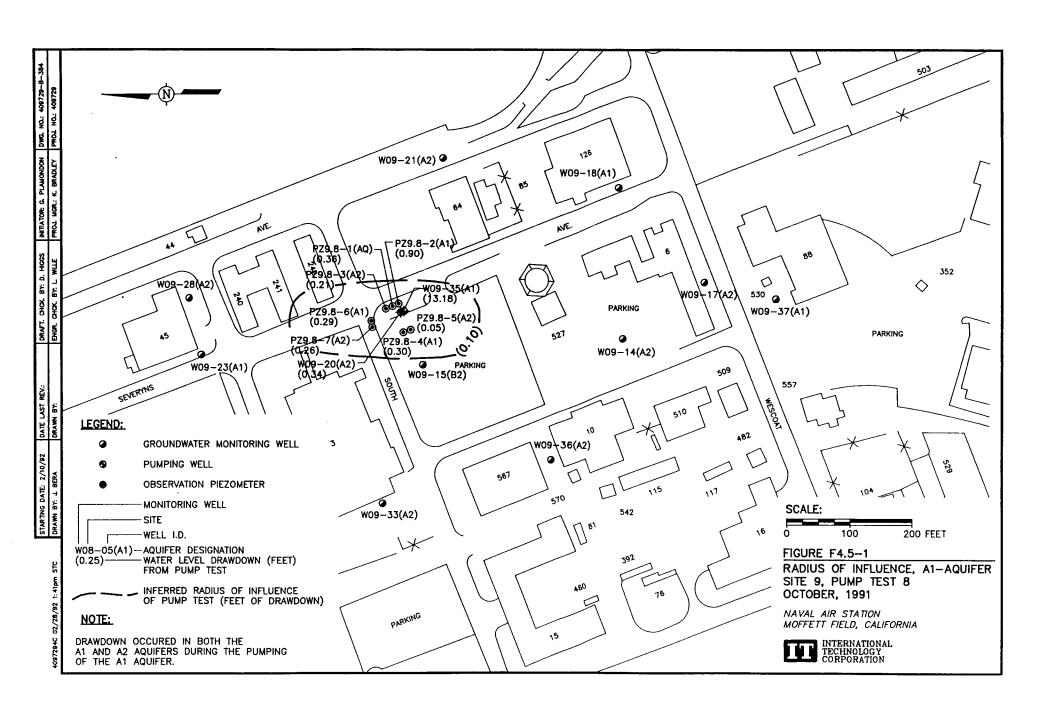


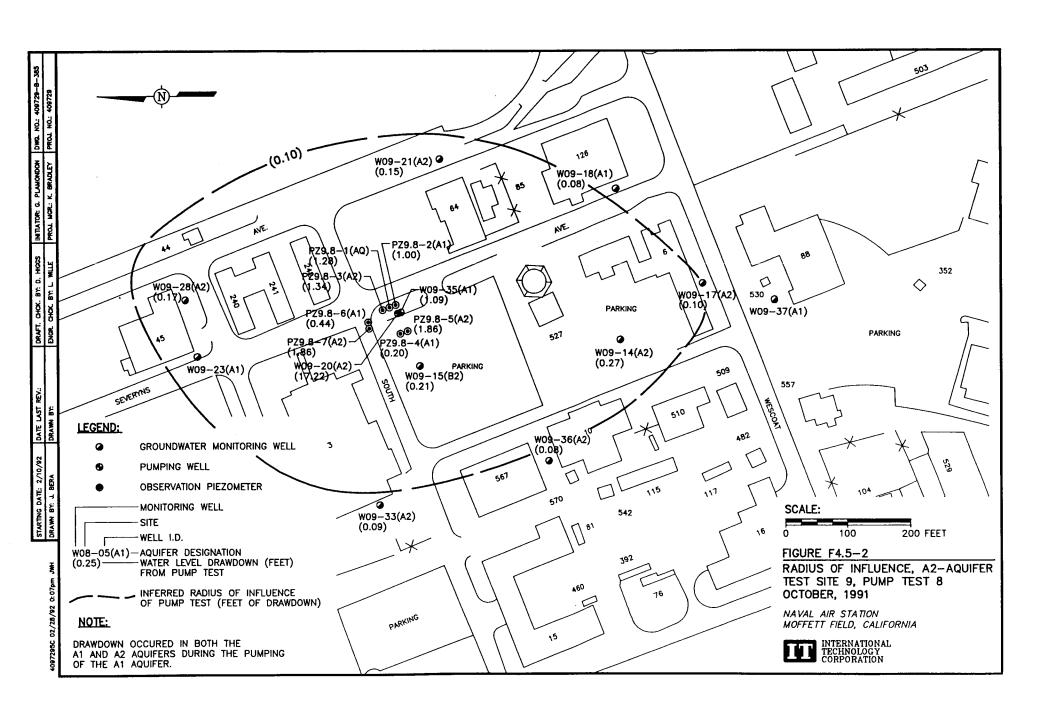
TECHNOLOGY CORPORATION

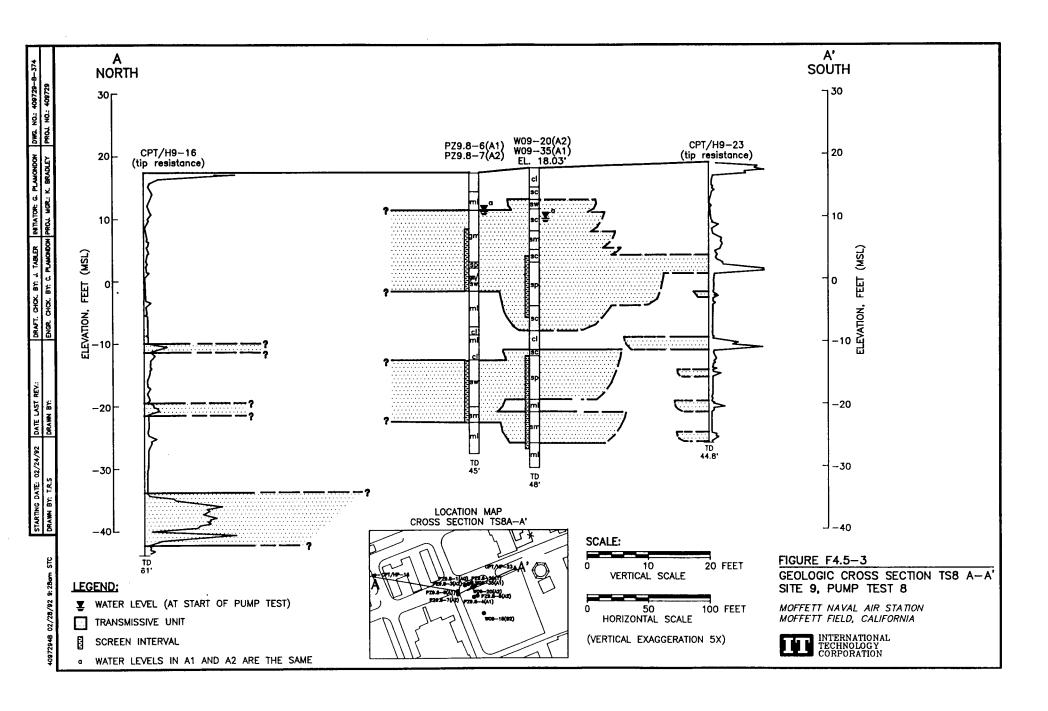
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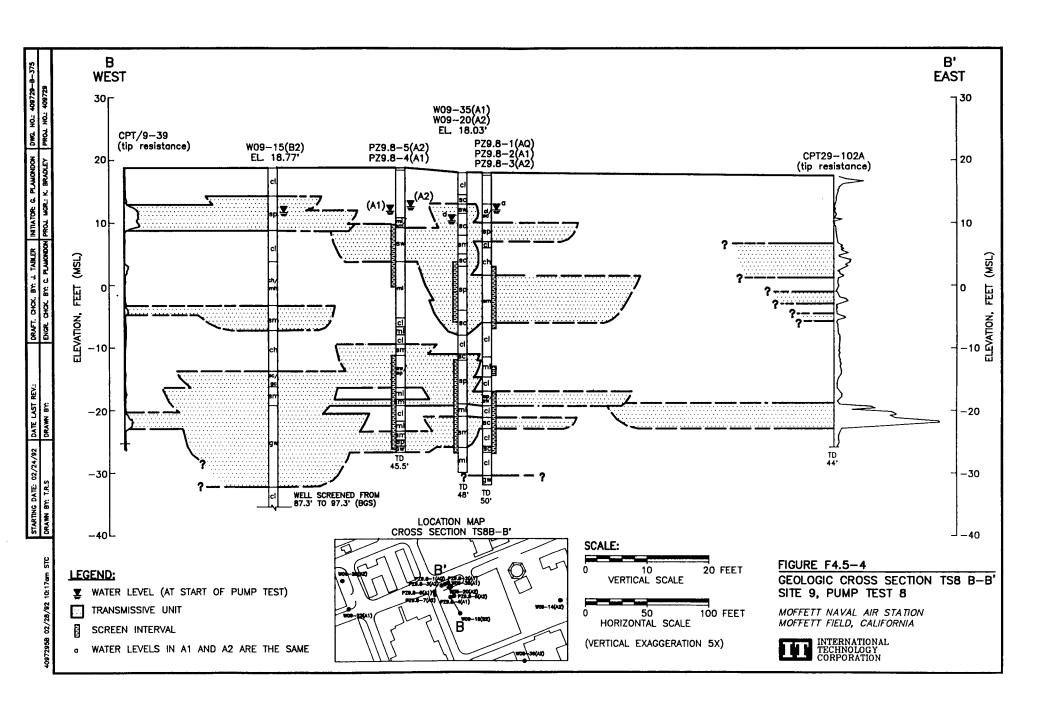


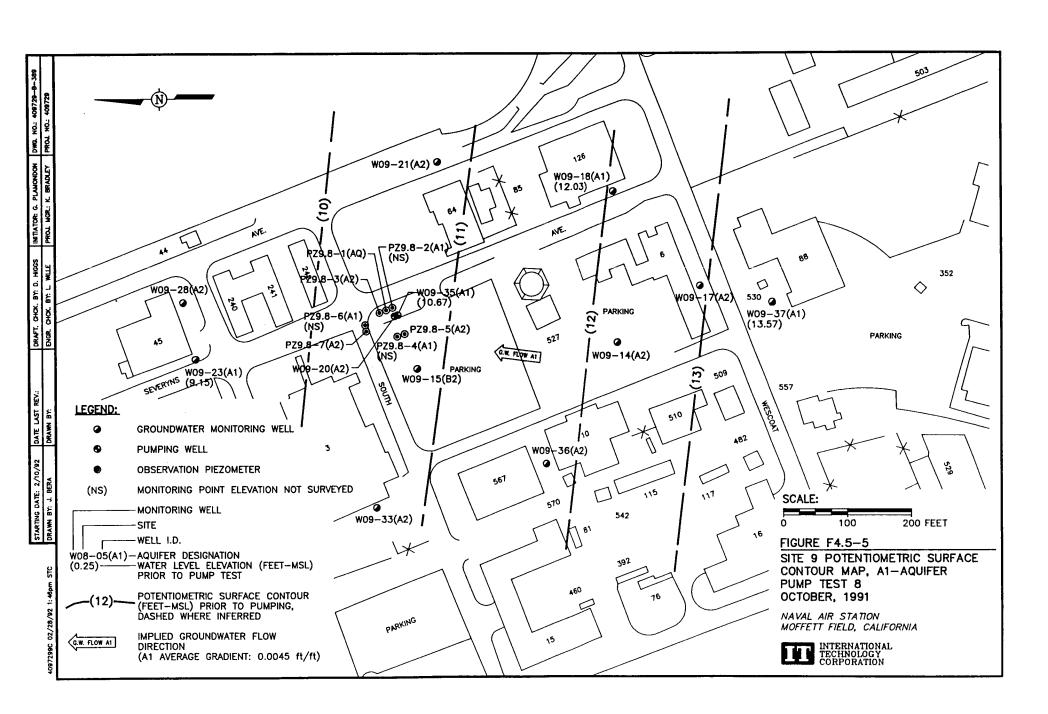


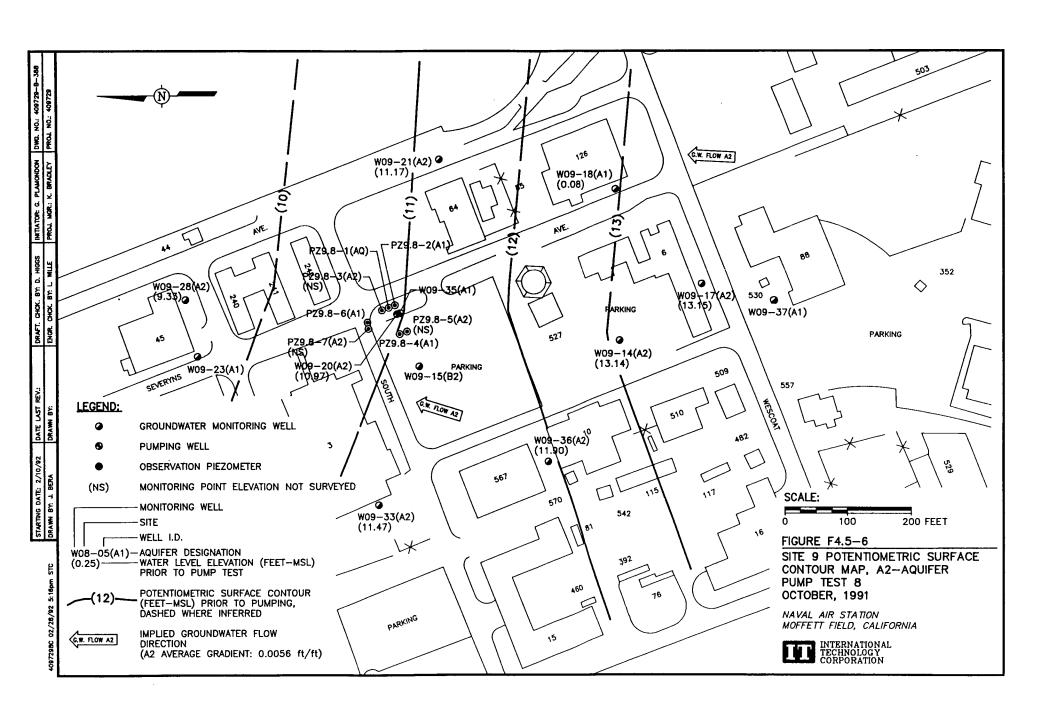


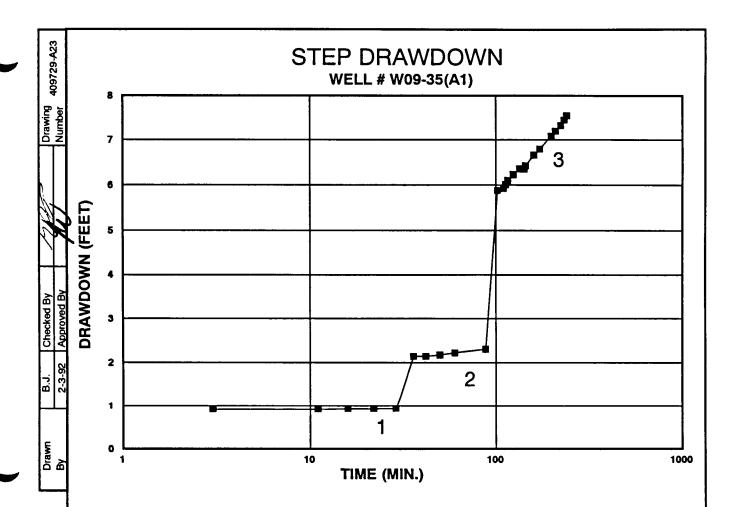












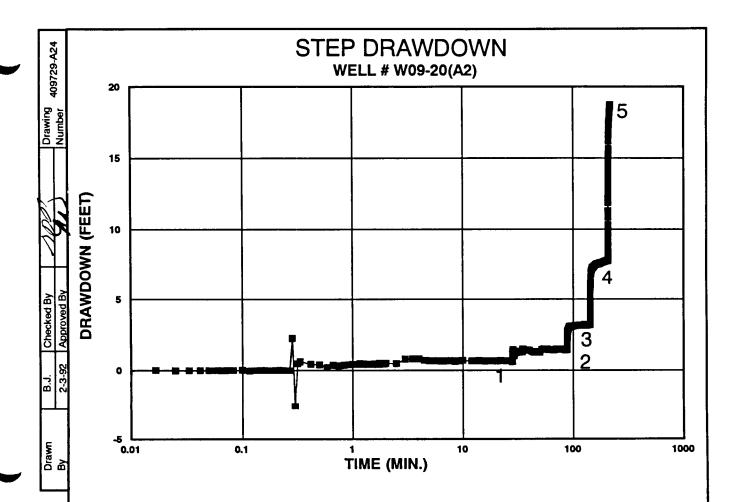
STEP#	DISCHARGE RATE Q (GPM)	CUMULATIVE DRAWDOWN s (feet)	SPECIFIC CAPACITY Q/s (gpm/ft)
1	1.1	0.95	1.158
2	2.4	2.35	1.021
3	5.7		NA

FIGURE F4.5-7 Pump Test 8 (A1)

Pump Test 8 (A1) Step-Drawdown Test

NAVAL AIR STATION MOFFET FIELD, CALIFORNIA





STEP#	DISCHARGE RATE Q (GPM)	CUMULATIVE DRAWDOWN s (feet)	SPECIFIC CAPACITY Q/s (gpm/ft)
1	1.4	0.7	2.000
2	3	1.5	2.000
3	6.1	3.25	1.877
4	12	8.25	1.455
5	23		

Note:

Data for the fifth step is invalid. Stabilization was not achieved due to encroachment on the well screen.

FIGURE F4.5-8

Pump Test 8 (A2) Step-Drawdown Test

NAVAL AIR STATION MOFFETT FIELD, CALIFORNIA



AQUITARD, A RESTRICTION INCORPORATING THE LEAKAGE FACTOR IS DICTATED BY:

 $tp + t' < (B^2S)/20T$ 

THEREFORE, VALUES OF T MAY BE OVERESTIMATED.

FIGURE F4.5-9 AQUIFER ANALYSIS THEIS RECOVERY METHOD

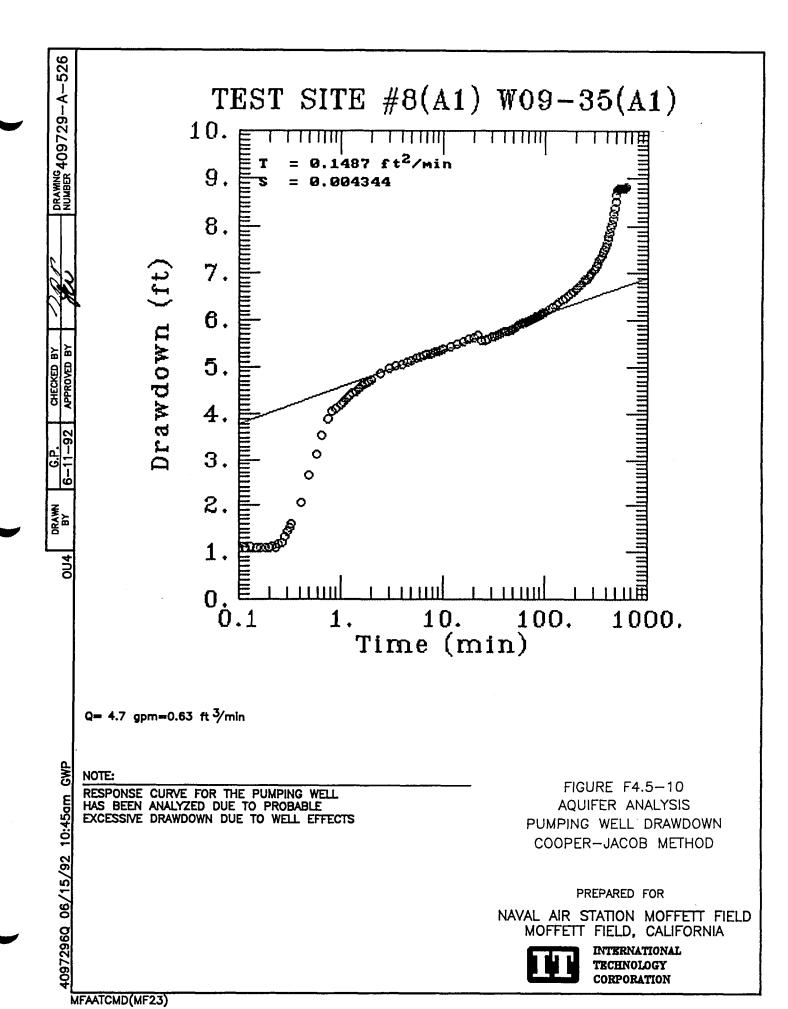
PREPARED FOR

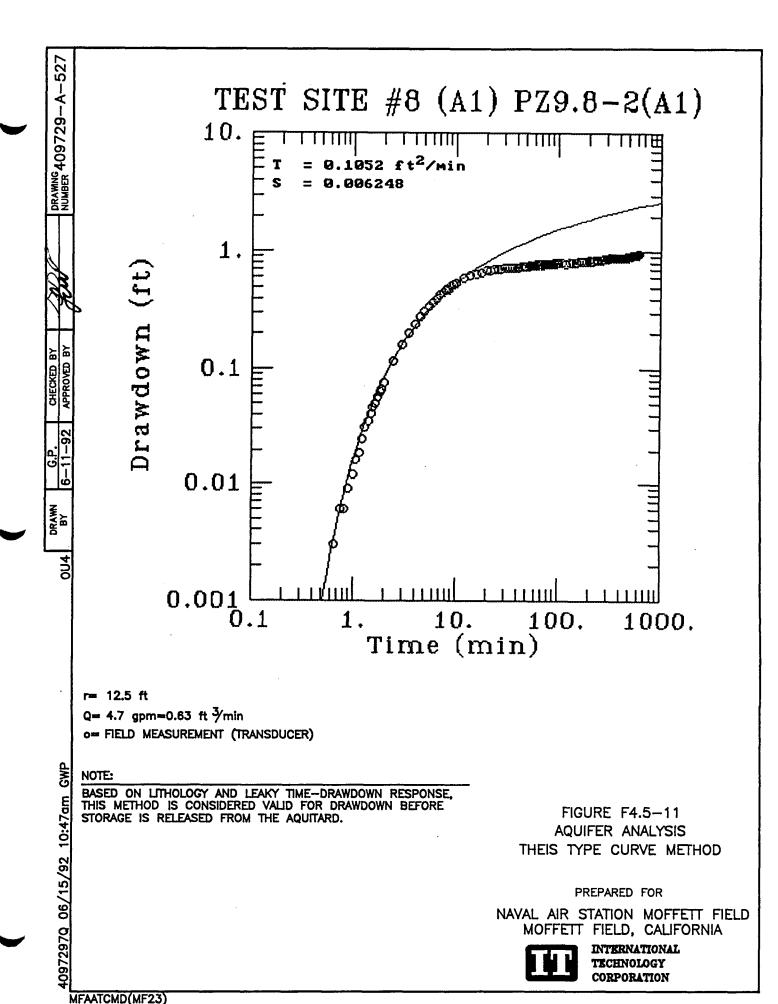
NAVAL AIR STATION MOFFETT FIELD MOFFETT FIELD, CALIFORNIA

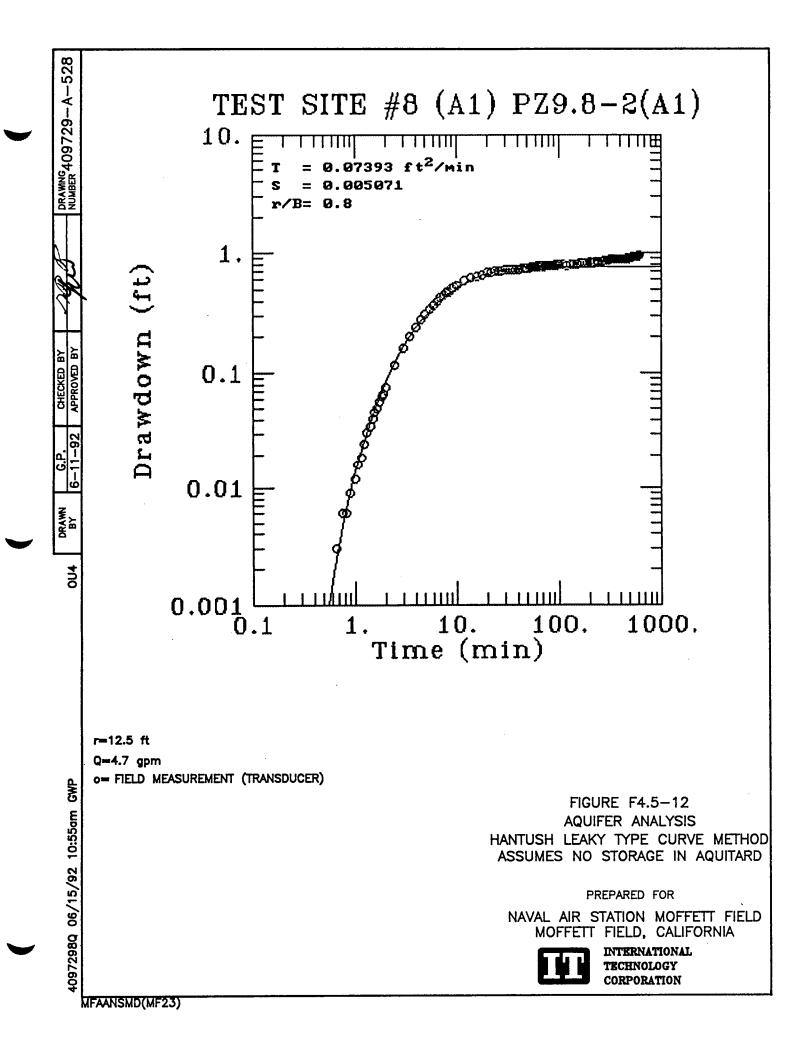


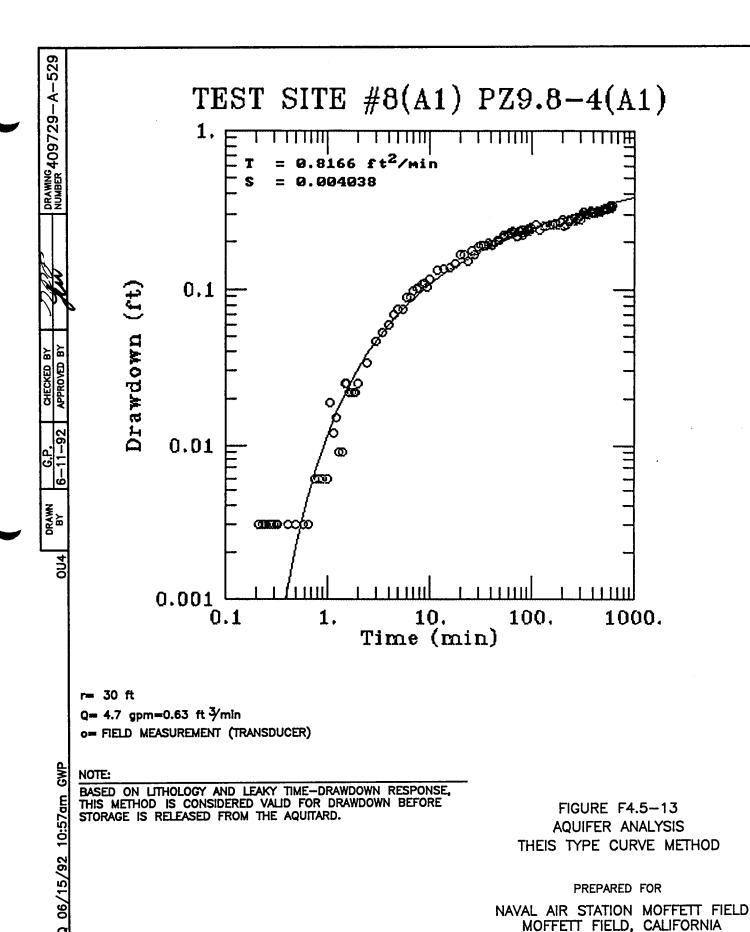
INTERNATIONAL TECHNOLOGY CORPORATION

4097295Q 06/15/92





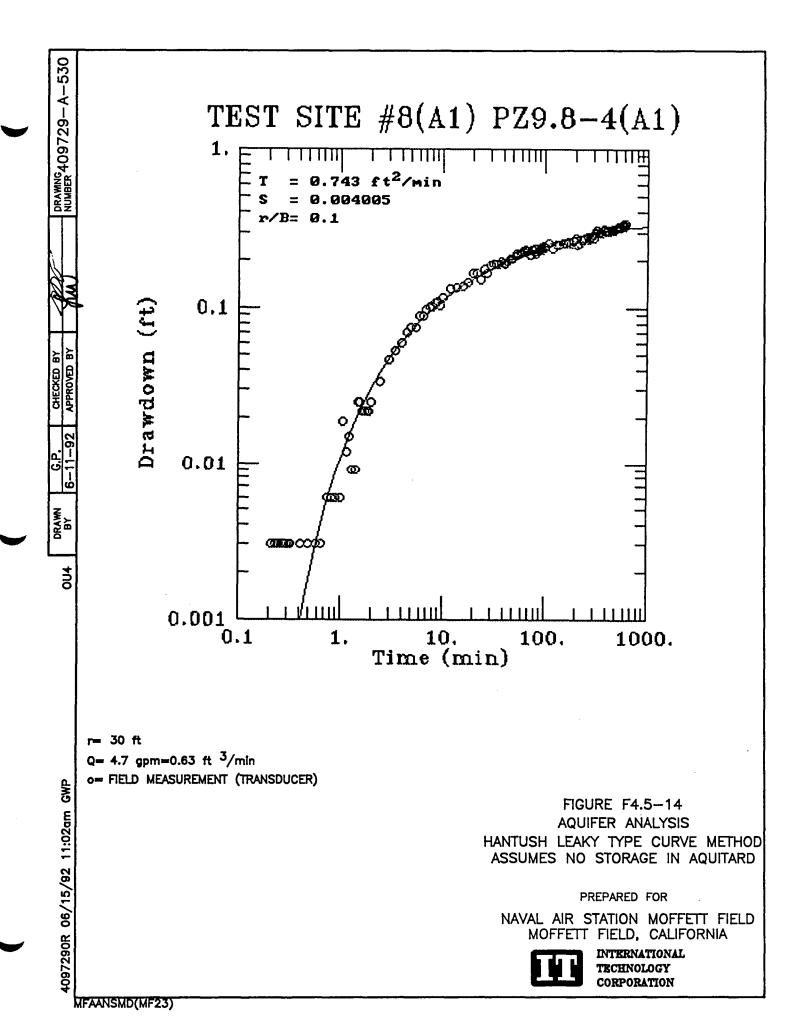


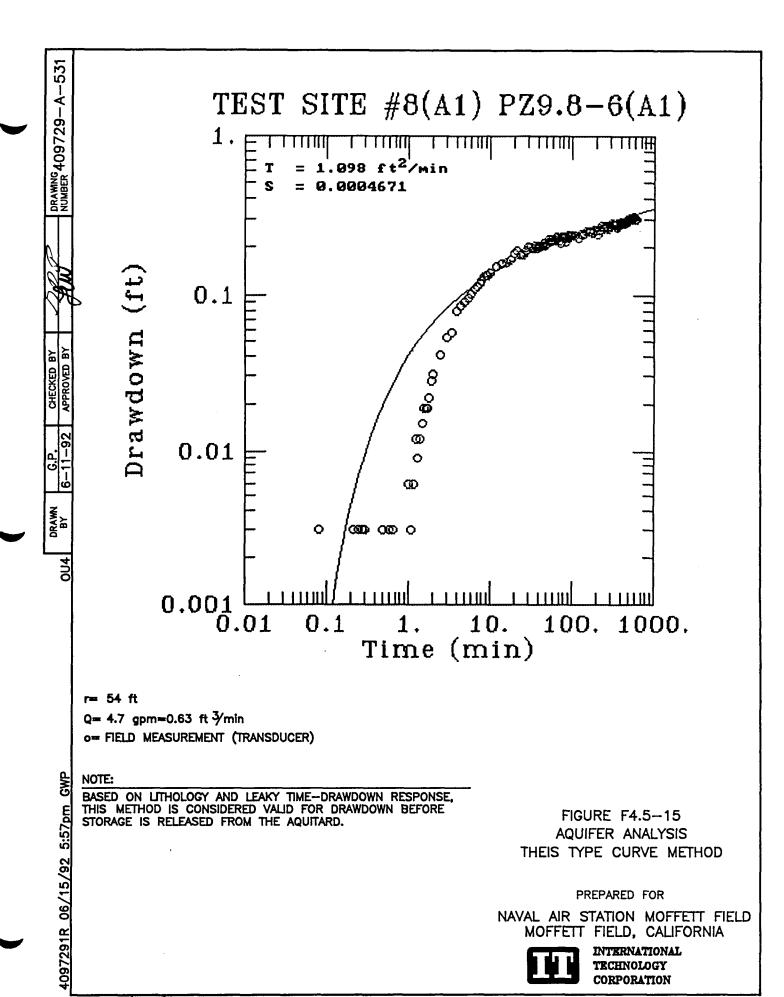


 $\mathbf{II}$ 

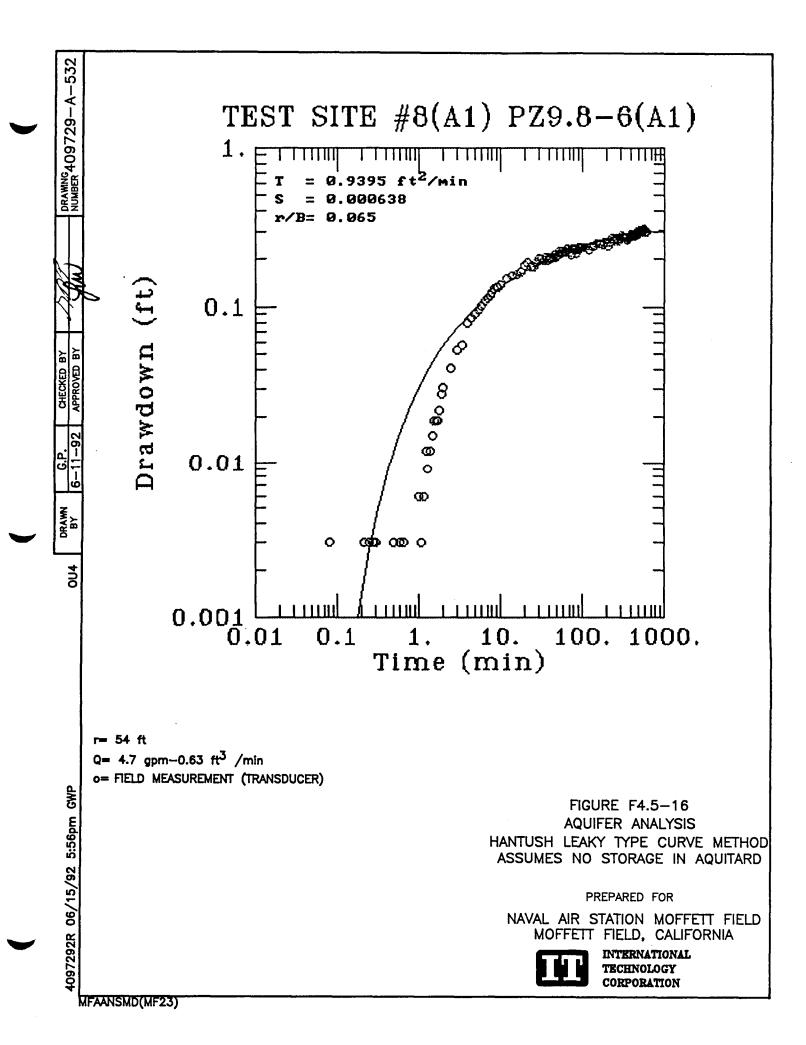
INTERNATIONAL TECHNOLOGY CORPORATION

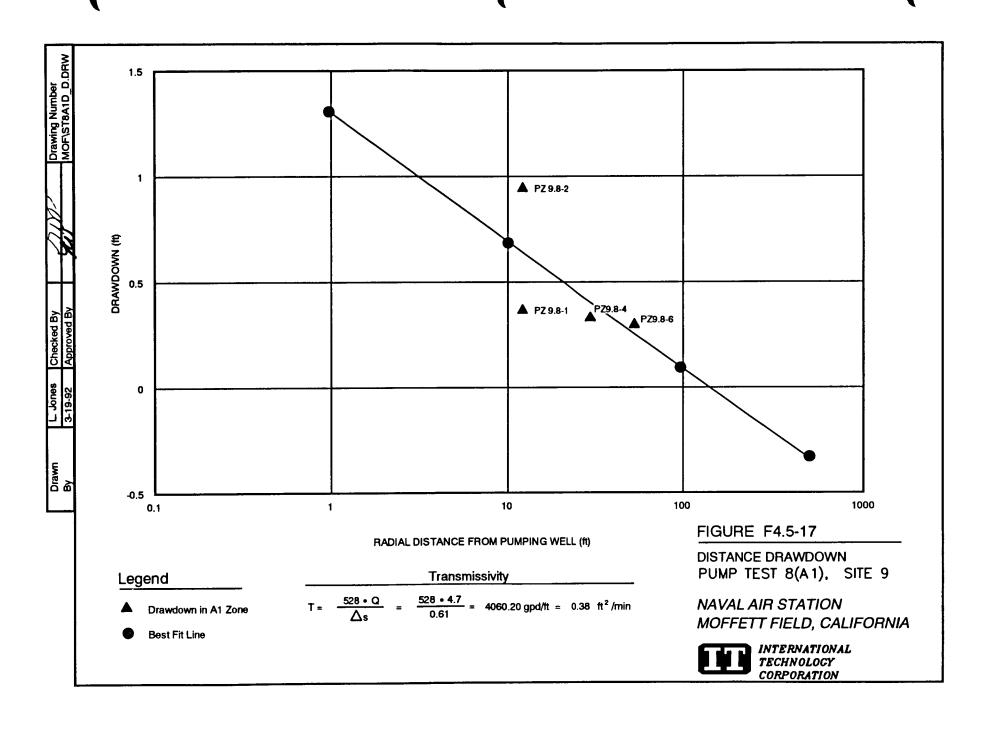
MFAATCMD(MF23)

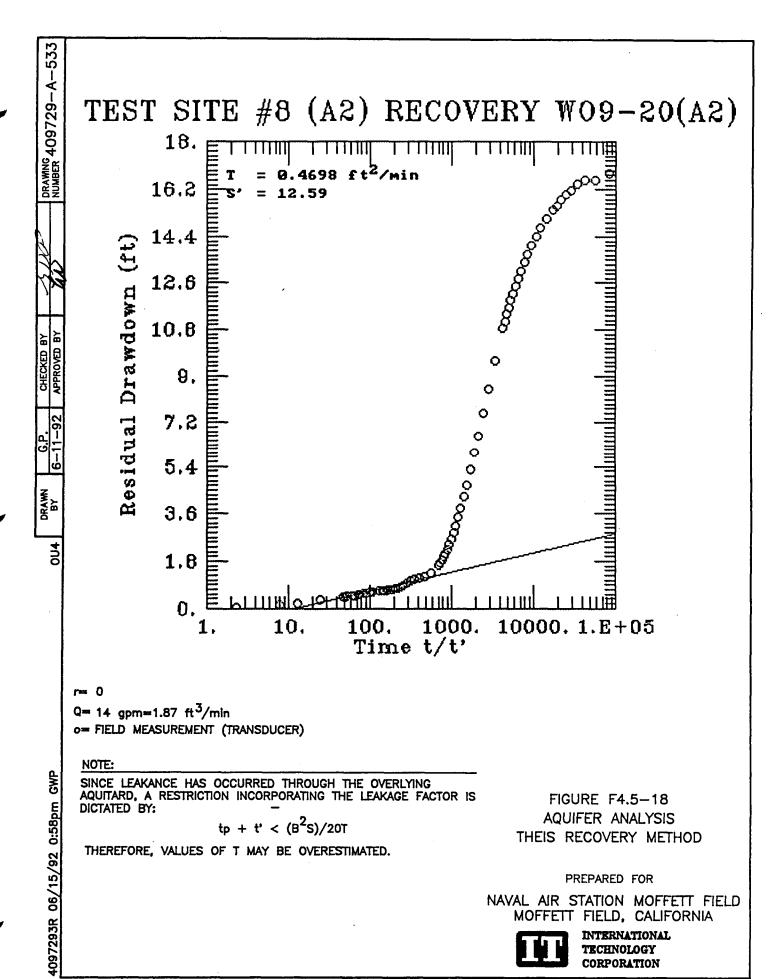




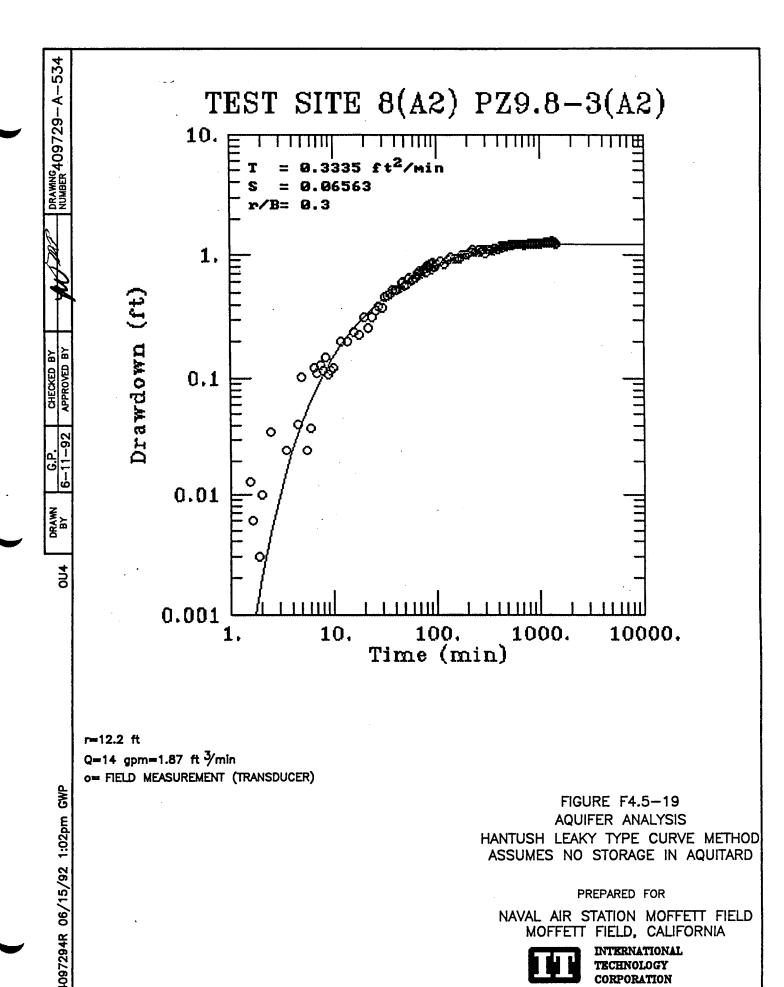
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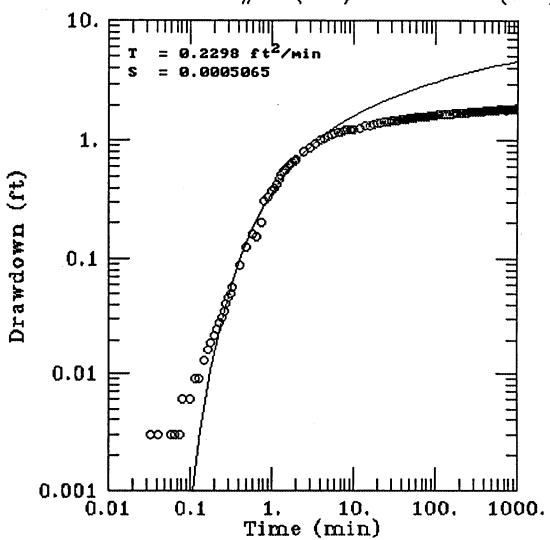


MFAATRMD(MF23)





## TEST SITE #8 (A2) PZ9.8-5(A2)



r= 30.2 ft

 $Q = 14 \text{ gpm} = 1.87 \text{ ft} \frac{3}{\text{min}}$ 

o= FIELD MEASUREMENT (TRANSDUCER)

#### NOTE:

BASED ON LITHOLOGY AND LEAKY TIME—DRAWDOWN RESPONSE, THIS METHOD IS CONSIDERED VALID FOR DRAWDOWN BEFORE STORAGE IS RELEASED FROM THE AQUITARD.

THEREFORE, DRAWDOWN DATA AFTER 4 MINUTES IS NEGLECTED IN THE CURVE MATCH.

FIGURE F4.5-20 AQUIFER ANALYSIS THEIS TYPE CURVE METHOD

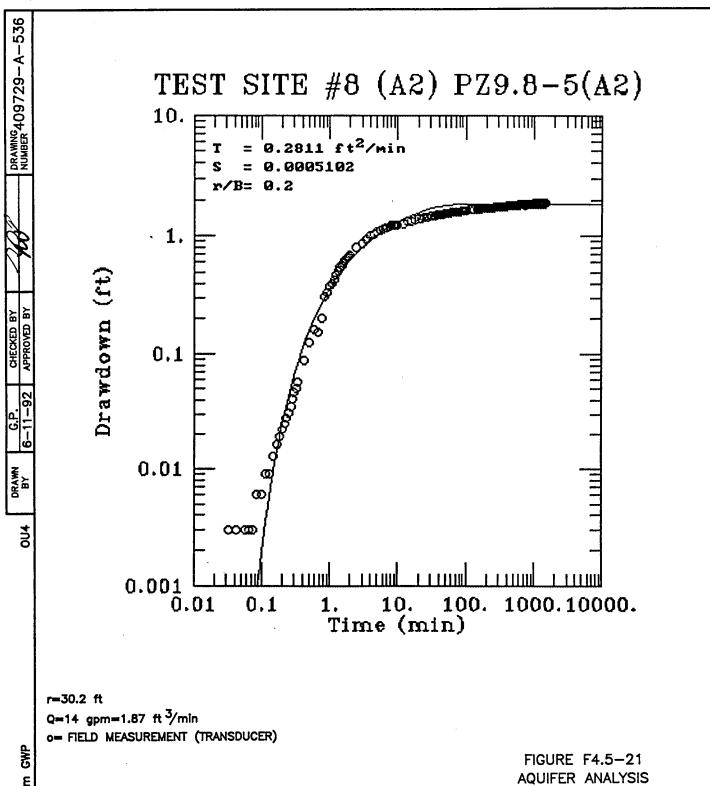
PREPARED FOR

NAVAL AIR STATION MOFFETT FIELD MOFFETT FIELD, CALIFORNIA



INTERNATIONAL TECHNOLOGY CORPORATION

295R 06/15/92 5:52pm G



HANTUSH LEAKY TYPE CURVE METHOD ASSUMES NO STORAGE IN AQUITARD

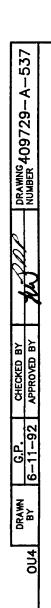
PREPARED FOR

NAVAL AIR STATION MOFFETT FIELD MOFFETT FIELD, CALIFORNIA

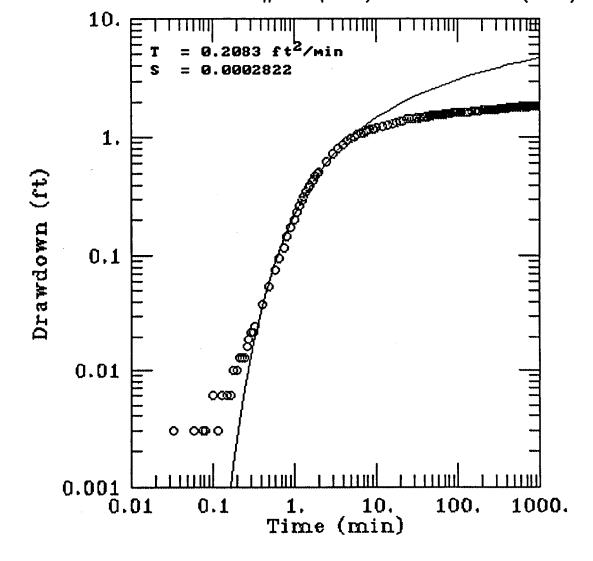


INTERNATIONAL TECHNOLOGY CORPORATION

4097296R 06/15/92 1:07pm GWP



### TEST SITE #8 (A2) PZ9.8-7(A2)



r=49 ft Q= 14 gpm=1.87 ft <sup>3</sup>/min o= FIELD MEASUREMENT (TRANSDUCER)

NOTE:

BASED ON LITHOLOGY AND LEAKY TIME—DRAWDOWN RESPONSE, THIS METHOD IS CONSIDERED VALID FOR DRAWDOWN BEFORE STORAGE IS RELEASED FROM THE AQUITARD.

FIGURE F4.5-22
AQUIFER ANALYSIS
THEIS TYPE CURVE METHOD

PREPARED FOR

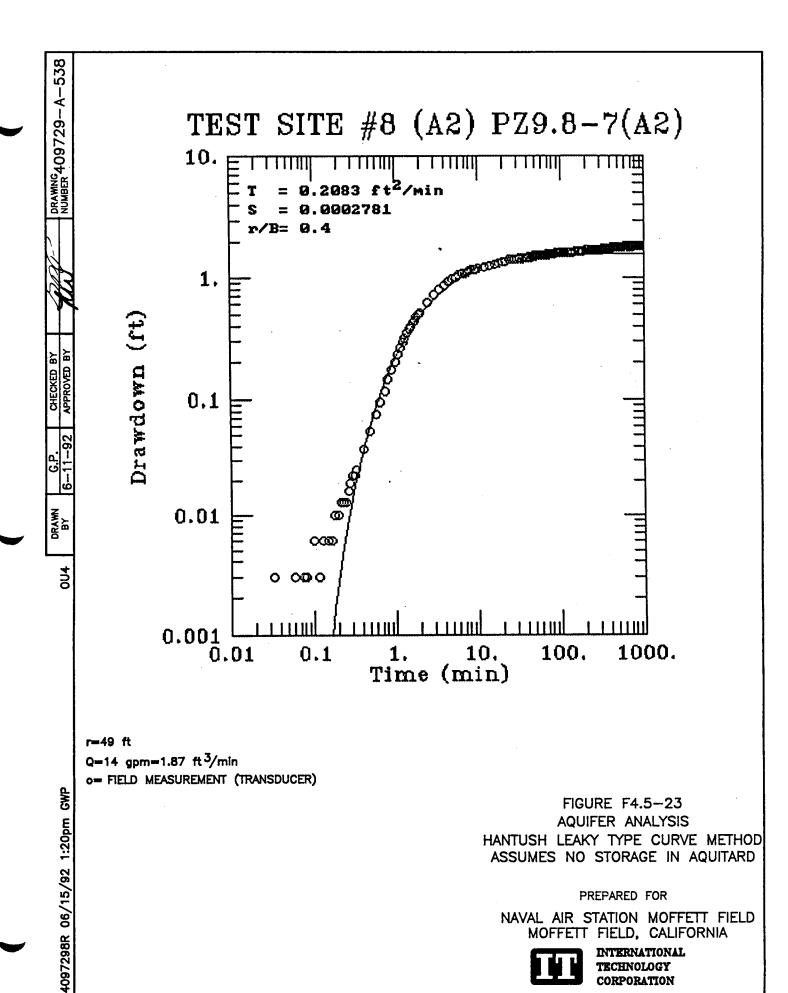
NAVAL AIR STATION MOFFETT FIELD MOFFETT FIELD, CALIFORNIA

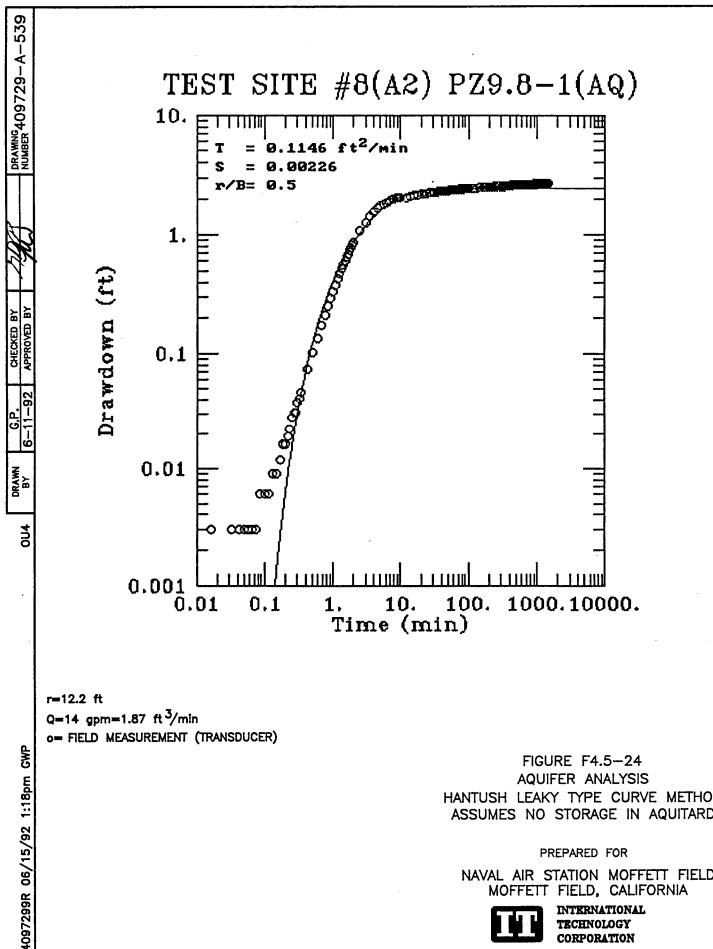


INTERNATIONAL TECHNOLOGY CORPORATION

7297P OF/15/92 1:14pm GWE

MFAATCMD(MF23)





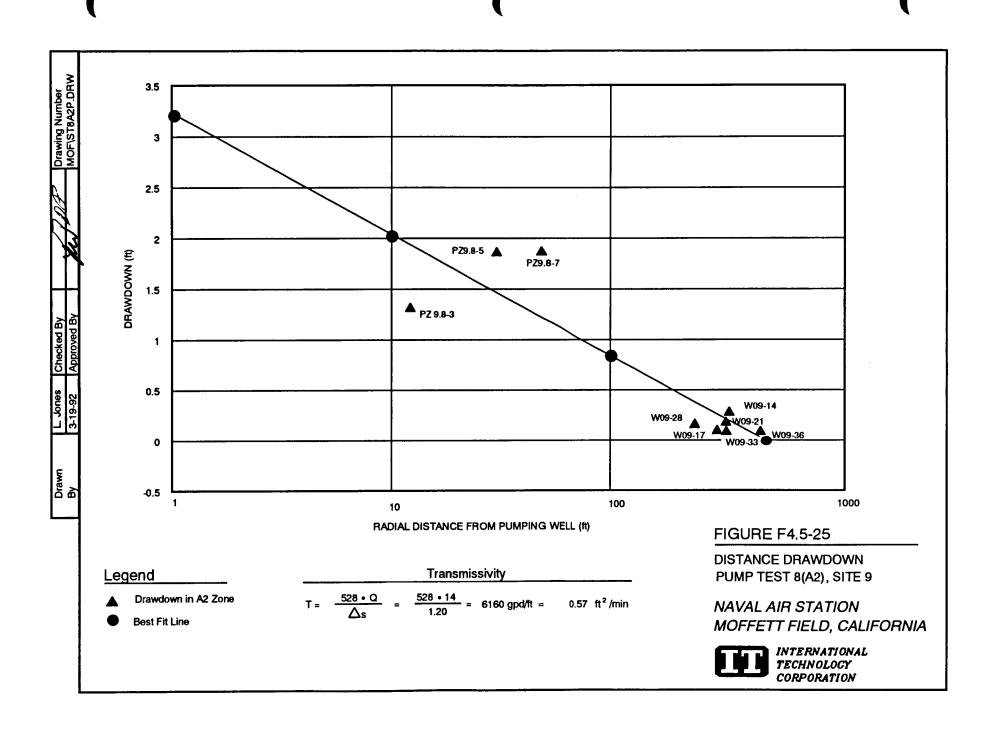
HANTUSH LEAKY TYPE CURVE METHOD ASSUMES NO STORAGE IN AQUITARD

PREPARED FOR

NAVAL AIR STATION MOFFETT FIELD MOFFETT FIELD, CALIFORNIA



INTERNATIONAL TECHNOLOGY CORPORATION



o= FIELD MEASUREMENT (TRANSDUCER)

USING A UNIT THICKNESS OF 11.0 FT. YIELDS A HORIZONTAL HYDRAULIC CONDUCTIVITY OF 0.00096 FT. 2/MIN.

FIGURE F4.5-26 SLUG TEST ANALYSIS COOPER, et al. METHOD

PREPARED FOR

NAVAL AIR STATION MOFFETT FIELD MOFFETT FIELD, CALIFORNIA



INTERNATIONAL TECHNOLOGY CORPORATION

1097290S 06/15/92 1:21pm G

MFA3(MF23)

ATTACHMENT I – PUMP TEST 7, SITE 8

THIS RECORD CONTAINS LARGE VOLUMES OF DATA AND IS NOT REQUIRED TO BE PHYSICALLY LOCATED WITH THE ADMINISTRATIVE RECORD DOCUMENT.

DUE TO EXTENSIVE VOLUME, THIS DATA WILL NOT BE IMAGED.

TO VIEW THE DATA, CONTACT:

DIANE C. SILVA
RECORDS MANAGEMENT SPECIALIST
NAVAL FACILITIES ENGINEERING COMMAND
SOUTHWEST
1220 PACIFIC HIGHWAY
SAN DIEGO, CA 92132

ATTACHMENT II - PUMP TEST 1, SITE 9

THIS RECORD CONTAINS LARGE VOLUMES OF DATA AND IS NOT REQUIRED TO BE PHYSICALLY LOCATED WITH THE ADMINISTRATIVE RECORD DOCUMENT.

DUE TO EXTENSIVE VOLUME, THIS DATA WILL NOT BE IMAGED.

TO VIEW THE DATA, CONTACT:

DIANE C. SILVA
RECORDS MANAGEMENT SPECIALIST
NAVAL FACILITIES ENGINEERING COMMAND
SOUTHWEST
1220 PACIFIC HIGHWAY
SAN DIEGO, CA 92132

ATTACHMENT III - PUMP TEST 3, SITE 9

THIS RECORD CONTAINS LARGE VOLUMES OF DATA AND IS NOT REQUIRED TO BE PHYSICALLY LOCATED WITH THE ADMINISTRATIVE RECORD DOCUMENT.

DUE TO EXTENSIVE VOLUME, THIS DATA WILL NOT BE IMAGED.

TO VIEW THE DATA, CONTACT:

DIANE C. SILVA
RECORDS MANAGEMENT SPECIALIST
NAVAL FACILITIES ENGINEERING COMMAND
SOUTHWEST
1220 PACIFIC HIGHWAY
SAN DIEGO, CA 92132

ATTACHMENT IV - PUMP TEST 5, SITE 9

THIS RECORD CONTAINS LARGE VOLUMES OF DATA AND IS NOT REQUIRED TO BE PHYSICALLY LOCATED WITH THE ADMINISTRATIVE RECORD DOCUMENT.

DUE TO EXTENSIVE VOLUME, THIS DATA WILL NOT BE IMAGED.

TO VIEW THE DATA, CONTACT:

DIANE C. SILVA
RECORDS MANAGEMENT SPECIALIST
NAVAL FACILITIES ENGINEERING COMMAND
SOUTHWEST
1220 PACIFIC HIGHWAY
SAN DIEGO, CA 92132

ATTACHMENT V – PUMP TEST 8, SITE 9

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DUE TO EXTENSIVE VOLUME, THIS DATA WILL NOT BE IMAGED.

TO VIEW THE DATA, CONTACT:

DIANE C. SILVA
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NAVAL FACILITIES ENGINEERING COMMAND
SOUTHWEST
1220 PACIFIC HIGHWAY
SAN DIEGO, CA 92132

ATTACHMENT VI - BORING LOGS

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DUE TO EXTENSIVE VOLUME, THIS DATA WILL NOT BE IMAGED.

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NAVAL FACILITIES ENGINEERING COMMAND
SOUTHWEST
1220 PACIFIC HIGHWAY
SAN DIEGO, CA 92132

ATTACHMENT VII – SOIL CONSOLIDATION TESTS

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DUE TO EXTENSIVE VOLUME, THIS DATA WILL NOT BE IMAGED.

TO VIEW THE DATA, CONTACT:

DIANE C. SILVA
RECORDS MANAGEMENT SPECIALIST
NAVAL FACILITIES ENGINEERING COMMAND
SOUTHWEST
1220 PACIFIC HIGHWAY
SAN DIEGO, CA 92132

ATTACHMENT VIII – GROUNDWATER
ANALYTICAL RESULTS

THIS RECORD CONTAINS LARGE VOLUMES OF DATA AND IS NOT REQUIRED TO BE PHYSICALLY LOCATED WITH THE ADMINISTRATIVE RECORD DOCUMENT.

DUE TO EXTENSIVE VOLUME, THIS DATA WILL NOT BE IMAGED.

TO VIEW THE DATA, CONTACT:

DIANE C. SILVA
RECORDS MANAGEMENT SPECIALIST
NAVAL FACILITIES ENGINEERING COMMAND
SOUTHWEST
1220 PACIFIC HIGHWAY
SAN DIEGO, CA 92132